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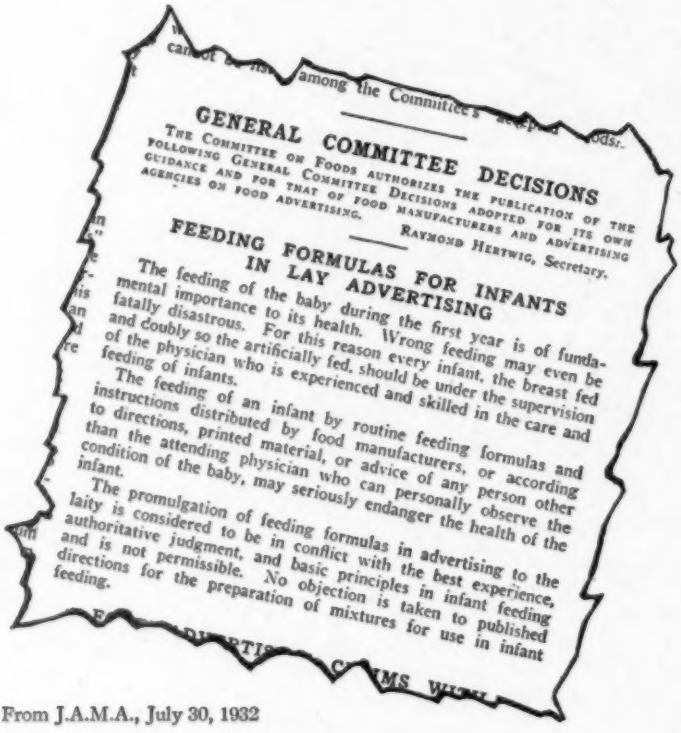

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From J.A.M.A., July 30, 1932

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The Journal of Pediatrics

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Original Communications

SCIENCE AND ART IN CHILD NOURISHMENT

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WINNETKA, ILL.

THE practice of medicine is as much art as science, and quite appropriately so, for art is never so convincing as it is when it is accurate, and science is never so pleasing as when it is artistic. The two concepts apparently so remote are, in reality, quite closely related as illustrated by the many great artists of scientific bent and the numerous scientists who have been artists as well. Emphasis here is placed, therefore, on the art of applying scientific advances to the growing members of the race. Need for the artistic touch in nutrition has been felt acutely since anorexia has become so common. Our frequent contacts with children and mothers, sometimes even with their fathers, give ample opportunity for the exercise of our talents along these lines.

Whereas in the usual discharge of our duties we act as thoroughly trained men whose word is law and is, more or less, implicitly obeyed, in dealing with children we find ourselves challenged by the fact that these little patients have no respect for our laws and often cannot be made to obey. Therefore, if we arbitrarily prescribe quantitative diets to children, we invite the possibility that the orders will be in part disobeyed. There is no *force* on earth which can overcome the refusal of a child to eat. At the sight of our dilemma, mothers, fathers, nurses, nutritionists, educators, psychologists, and, it seems, most of the world gather about to give advice, often with a touch of satisfaction in our discomfiture. If we are to maintain our prestige as competent advisers in the matter of children's diets, it becomes necessary that we tell mothers not only what to feed them, but also how to insure the actual ingestion of food.

The problem thus presented takes us out of the purely physical field, if there is any such thing, and forces us to consider the mental or

Read before the Academy of Medicine, Cleveland, May 20, 1932.

psychologic factors involved in this refusal of food. In this unaccustomed field of activity, we are at once given an opportunity for cooperative study with workers in allied fields dealing with the behavior of children, and are given the serious responsibility of arriving at common sense conclusions in harmony with the best thought of the time. We find ourselves associated with men and women who know more about certain aspects of the subject than we do, a somewhat novel experience. In making suggestions, therefore, an effort is made to see that they are harmonious with the ideals of progressive thought on growth and development both physical and mental.

Lest any one think that I do not care what children eat, I should like to state at the outset that all that is said has but a single purpose. This is to make available to all children the benefits of recent brilliant discoveries in nutrition, such as the influence of amino acids, vitamins, mineral salts, and the water balance on growth and health. These discoveries will go down in history among the greatest of scientific accomplishments. To apply them to the present generation is our task.

In many ways the subject of nutrition is the most vital one affecting the human race today. We are all living a tremendous drama, the struggle of a scientific civilization against the fixed laws of nature, many of which we openly contest. In less enlightened days, when the law of the survival of the fittest held, only the strong and hardy survived and handed down their vigor to succeeding generations. By saving the lives of puny children, we now allow weaklings to mature and add their less stalwart strain to the germ plasm of the race. In doing this, medical science challenges itself so to improve the growing race that it will in some measure compensate for these self-imposed defects—otherwise the race must of necessity deteriorate physically. Nutrition obviously plays a leading rôle in this effort at individual and race improvement.

The roots of the anorexia problem go back to the time when the physiologist discovered the fact that the food calory was a mathematic entity. At that time, many workers jumped to the conclusion that the calory was *the* unit of nutrition and therefore urged that children be given high caloric feedings. They implied that such high caloric feedings were necessary for normal development and successfully popularized the idea. We now know that this is far from the truth. The calory is a unit of *combustion*, not of nutrition. However, under the stimulus of this idea, we saw excessive development of the foods yielding the most calories at the least effort and expense, the carbohydrates. Numberless breakfast foods appeared on the market like magic, puddings were elevated from their former dubious position and extolled as bearers of bright and shining calories. It was maintained that armies could march untold miles on cakes of chocolate, and blatant advertising of the day capitalized the calory much as it now does the vitamin, and

with less justice. Since gain in weight often followed restoration of health after illness, it was quite naturally assumed that weight increase was an index of good health—witness the photographs of prodigiously fat and rickety babies illustrating various infant foods. Adiposity became synonymous with health. With this development, we come to the height of what might be termed the quantitative era in nutrition, quantitative in regard to both caloric intake and human flesh.

From the appetite standpoint, the important early result of this feverish activity was the fact that calories were so conscientiously and thoughtlessly crammed that the appetites of a surprisingly large proportion of our children were ruined. Caloric propaganda still wields considerable force throughout the country. Even now, when every new discovery seems to emphasize the quality factor in feeding, when inconceivably small amounts of unknown substances are shown to be necessary for life itself, we still hear slogans such as "A quart of milk a day," and see parents fearful that malnutrition will overtake their children if they do not force them to eat some prescribed amount of this, that, or the other food, and we find health estimated on the basis of avoirdupois.

Lately, the light of modern scientific discovery has been steadily melting away this idol of fat. Realization of the importance of certain amino acids in the very maintenance of life challenged the caloric idea. Then came Funk, Hopkins, Osborne, Mendel, McCallum, and others with their still more startling discoveries of the vitamins, called so because it was at first thought that they were amino acids, or their derivatives. Without these, specific diseases resulted and often life itself could not exist. One after another new essential factor in nutrition has flashed on the scene, so many in fact, that today the bewildered physician trembles when he opens his daily mail for fear that it will bear tidings of some new vitamin or anti-anemia salt, to tax his memory. In this vitamin-mineral-salt era our friends the investigators have set our thoughts jumping back and forth from iron to copper, iodine to manganese, haliver oil to yeast, etc., so rapidly, that it is next to impossible to correlate intelligently the mass of scientific facts put at our disposal. And then, as a crowning challenge to our equanimity, we are obliged to answer more or less intelligent questions on this subject from our patients before the facts are established. Nevertheless, we cannot shirk the situation which demands our attention and challenges our vision.

We must realize that we are at present living in a period of such rapid change that it is hard to predict our dietary prescriptions from month to month. What are we likely to think of our present-day régime ten years from now? I imply that today the wise clinician will try to select such foods for his patients as will contain the food essen-

tials which the investigators of tomorrow are going to discover. Judging from past experience one can state unhesitatingly that such diets will consist in widely diversified selections of foods as little changed by manufacture, seasoning and cooking as possible. Such diets have produced the race and if properly supplemented should sustain it. Undoubtedly specific substances of proved value will be added from time to time to both individual and community diets.

Such a progressive program is dictated by common sense and prudence, and its achievement is blocked by one factor only, widespread anorexia in children. This, it is suggested, is due to forced feeding initiated by a misconception of the very fundamentals of nutrition, a hand-me-down from the caloric or quantitative era. You will note that even the youngsters rebelled at being subjected to so unscientific a procedure as forced feeding. As a result, unfortunately in many instances, neither quantity nor quality is eaten, and the whole nutritional system breaks down; family strife, unhappiness, behavior problems, phobias, and indigestion appear.

We are "all set with no place to go" unless we can control this problem of the appetites of our young patients. The development of good appetite, an attribute which is well established during the first few months of life, becomes, therefore, a major objective in nutrition. Appetite is that which makes us think pleasantly of food and which makes us continue to eat because we enjoy it. Hunger is an uncomfortable sensation in the abdomen caused by vigorous contractions of the empty stomach and is allayed by the ingestion of anything. Appetite is a psychic function, hunger a physical one. It is highly probable that these two in some way, alone or aided by some undiscovered center in the brain, form a mechanism which automatically controls the amount and kind of nourishment taken. It is hardly conceivable that the race could have developed in the absence of such a mechanism any more than it could have lived without the carbon dioxide control of pulmonary ventilation. Dr. Clara Davis in her experiments on the self-selected diet of infants has indicated the existence of some mechanism, for each infant chose, month in and month out, foods which totaled within 1 per cent of the same proportion of fat, carbohydrate and protein. Each infant, moreover, chose his own proportion, and having done so, stuck to it in spite of occasional egg, banana, and meat jaggs. If this mechanism exists it must be intimately tied up with a feeling of well-being, because all through the ages good times have been associated with eating. When we study methods to further good appetite, then, we are really undertaking a worth-while job; when we tamper with it, we are treading on dangerous ground, not only from the standpoint of nutrition, but also from that of the happiness of the individual.

As far as we know today, the attributes of the newly born infant which enables him to eat adequately are: (1) hunger contractions which

course over his empty stomach; (2) the ability to cry when made uncomfortable; (3) the coordinated reflexes having to do with sucking and swallowing; (4) his ability to react pleasurably toward his environment; and (5) the presence of a mother or nurse. A study of the mechanism by which these contribute to his nourishment may indicate something of the normal early control of eating. If the resulting hypothesis seems reasonable, we may be justified in adopting it until the physiologist gives us a more detailed one based on experiment.

Let us consider the beginning of appetite and trace its development through the vicissitudes of infantile life. The infant comes into the world in a most uncomfortable manner, to him as well as to his mother. He is squeezed, hauled about, plunged into a cold atmosphere, and dazzled by light. To this veritable bombardment of unpleasant stimuli he responds, unless weak or seriously injured, with a lusty cry of protest. Subsequently he is warmed, soft things are wrapped around him, the room is darkened, and he lapses back into a semblance of his sensationless intrauterine existence. Out of this void the baby is disturbed by one of the few early stimuli initiated in his own body, the hunger contraction. The result is that almost the earliest unpleasant sensation after delivery is abdominal distress due to hunger, manifested by the cry. In a strictly natural environment this cry leads to feeding with the breast or bottle. In either event the baby's nursing reflex is brought into play, and liquid finds its way into his stomach. With the first swallow the abdominal distress is relieved because hunger contractions cease when anything is introduced into the stomach. In this first experience with food the baby is comforted, and in his pleasurable reaction we may see the beginning of his good appetite. With frequent repetition of this experience registering itself over and over on the relatively clean slate of the brain's receptive centers, it becomes easy to see why the normal child grows up with a passion for eating. It is almost the earliest pleasant sensation and it is the one most frequently repeated.

If we accept this pain-relief mechanism as the one upon which good appetite is developed, our first consideration should be to establish a feeding technic which will foster it or at least not interfere with it. We must treat this natural reflex with the same respect we are accustomed to give to other manifestations of growth and development. Yet, it is obvious that even in its tenderest and most vital birth period, the growing appetite is ignored in our best hospitals. Indeed, the fact that all appetites are not ruined during the infant's first two weeks is proof of the adequacy of nature's provision. Babies are taken from their warm cribs by the clock, not when their own discomfort arouses them. If sleep resists this rude awakening, they are aroused sometimes by spanking, snapping the feet, or by dashes of cold water, and then are

expected to enjoy the meal—often before there is any meal to be enjoyed. The arroganee of adults toward children has no better illustration than such behavior toward the newly born infant. In this we find an adequate etiologic factor for *anorexia nervosa neonatorum*. The obvious times for the first nursings are when the baby cries, not at 6—10—2—6—10, etc. As soon as he learns to nurse, the hours may be adjusted to our convenience. The fact that such anorexia of the newly born is seldom seen where hygienic conditions are poor and people are ignorant is evidence that our hospital technics should be modified in this regard.

If the appetite of the infant has jumped this early hurdle and has emerged as a functioning mechanism, and if food is not urged to an unpleasant degree, there should be little difficulty during the next few months on breast milk or modern rather concentrated formulas. The greatest danger to appetite at this time lies in the enthusiastic nurse or mother who likes to boast of her child's remarkable gain and with that end in view urges food unduly. Here again adult arrogance threatens a vital function, innocently it is true, but also ignorantly. We have strong presumptive evidence that even at this early age the appetite of a child is an adequate index to the amount of food necessary. If gain in weight is unsatisfactory, it is much better practice to increase the concentration of the formula than to try to force a greater volume than the child desires.

Every breast-fed baby should be educated into the technic of bottle feeding from birth because such treatment prevents weaning difficulties. After the child has learned to suck from the bottle, it should be offered him once or twice a week, but he need not be made to take large amounts in this way. It is emphasized that the bottle is *not* given at this time as a major source of nourishment but is a purely educational instrument. I consider this one of the most important prophylactic measures in the field of child nurture.

At some time during the first few months, it is customary to introduce cod liver oil and orange juice into the diet. It is probably best to begin cod liver oil in the first weeks because it is not pleasant to the educated taste and usually is accepted well in the early days. Orange juice may be given soon afterward, and its introduction, if it is not too sour, causes little trouble.

The first radical change in the technic of feeding comes at the time when it is thought desirable to begin with the spoon, about the fourth or fifth month. This also should be approached as an educational effort of first magnitude rather than as a means of introducing nourishment. The spoon, with a small amount of some cereal mixed with his old familiar formula should be offered before the bottle or breast. No surprise should be felt if at first he does not discover that eating in this way is just as pleasant as was his old method. Usually, however, after

a few kindly offerings he gladly accepts the spoon. If not, the attempts should be gently continued or postponed for a week or ten days. In my experience the addition of sugar to such foods as cereals has in the long run retarded appetite rather than helped it. It is never advised. In any event, the baby should not be wrapped in a blanket, hog-tied and choked into spoon eating. As soon as the infant learns to take considerable amounts from the spoon with pleasure, and not before, this method may be used as a contributory factor in nourishment. Prior to this time it is an educational project. A four-month-old so treated should have a good appetite for milk, cereal, orange juice and cod liver oil and should know how to swallow food from the breast, bottle, or spoon.

The next new taste is called out when vegetables or purées are added, a month or so later. The same technic is employed as with cereals, and it is usually easier since by this time the spoon is recognized as the bearer of good tidings. A few years ago I discovered, partly from observing Dr. Davis' babies and partly by accident, that infants seem to enjoy raw grated vegetables much better than they do the cooked ones, and since that time these have been offered to all of my babies. The vegetable problem has disappeared with this treatment as the infants "hoe into" these raw foods as though made for them and rarely choose the cooked vegetables. It is with regret that I record that spinach has largely disappeared from the diet list, as no mother can grate it. Raw vegetables properly grated have proved less laxative than cooked vegetables according to the evidence of practically all mothers and nurses who have had experience in such feeding.

The introduction of baked potato is often met with opposition, probably because of its mealy consistency. Where this objection is persistent it may be well to substitute rice or spaghetti, since the food value is practically the same, and the consistency seems more pleasing.

Meat eating is the next educational step, usually attempted at eight to ten months. Raw or slightly cooked scraped beef is almost always eagerly taken by the child who has been well treated up to this time. If, on the other hand, he refuses, it is a matter of little importance because by this time the diet is so varied that adequate nourishment without meat is possible.

One more important step must be undertaken in the latter part of the first year, and that is the feeding of more coarsely divided food. This change should be made gradually as some babies will choke and gag when large particles first appear; it should also be made relatively early as infants accept the change better before they are too long accustomed to purées exclusively. In more or less rapid succession we can now give fruits, desserts, eggs, bacon, and toast so that by the time the youngster has passed his first birthday all of the important food elements are well received.

The only adult feeding trait the child has not learned by this time is that of feeding himself. To teach this, one has merely to place an extra spoon on the tray and let nature take its course. It may be hard on the wall paper and carpets, but it usually is not long before most of the food finds its way into the stomach on the baby's own power. Once the child has learned this, the attendant's duties are simple. They consist merely in serving the food and in getting out of appetite's way. Many a good start is spoiled by tampering with the child's inclinations after feeding habits have been well established.

No discussion of this subject will be complete without a word as to the treatment of the many early attacks of physiologic anorexia which complicate the management of the feeding régime. Whether the cause of the refusal to eat is an upper respiratory infection or an abdominal upset, the treatment should be the same, immediate, sharp reduction in the amount of food offered. This may be accomplished by skimming the milk, or by actual reduction in bulk, or both. If such steps are taken and the child is not urged, when the cause of the physiologic anorexia is removed, appetite will return vigorously and make up for lost time. If, as often happens, the ill child is pestered, spanked, and forced at feeding times, he will emerge from his attack with ruined appetite as a scar. The majority of patients with chronic anorexia date its onset from some such experience.

A word must be said in defense of the good but small appetite. Many children of small stature naturally require less food. They eat well for a while and then stop. Such behavior is natural and physiologically adequate for the nourishment of their smaller bodies. The trouble is that it does not satisfy our adult ambition for the bigger and better baby. These unfortunate inherently small individuals are usually crammed and forced until all semblance of appetite is educated out of them.

The foregoing technic has been advocated from a theoretic standpoint based upon what we know of the development of the eating urge. Its practical value depends upon its success first, in securing adequate nutrition, and second, in avoiding the condition known as chronic anorexia. Several years ago I reported a series of 200 consecutive cases in which I found that with a similar technic it was possible, even at that time when forced feeding was the approved method, to reduce the reluctant eaters to 15 per cent of the total and to obtain average weights 3.6 pounds above the normal for age. I have recently tabulated a consecutive group of 125 babies from private practice, charting birth weights against year-old weights. The accompanying scatter diagram (Fig. 1) illustrates the result. It will be seen that very few babies were below average weight and that the few anorexia cases were in this

group, designated "a." In most instances these smaller infants were unduly urged in spite of my advice, giving rise to the 7 per cent who are now "feeding problems."

In evaluating this, it must be remembered that all patients do not obey orders implicitly and that, therefore, these results may be said to follow when the physician gives the above instructions, not when the baby is so treated. I know that many of the failures have been due to

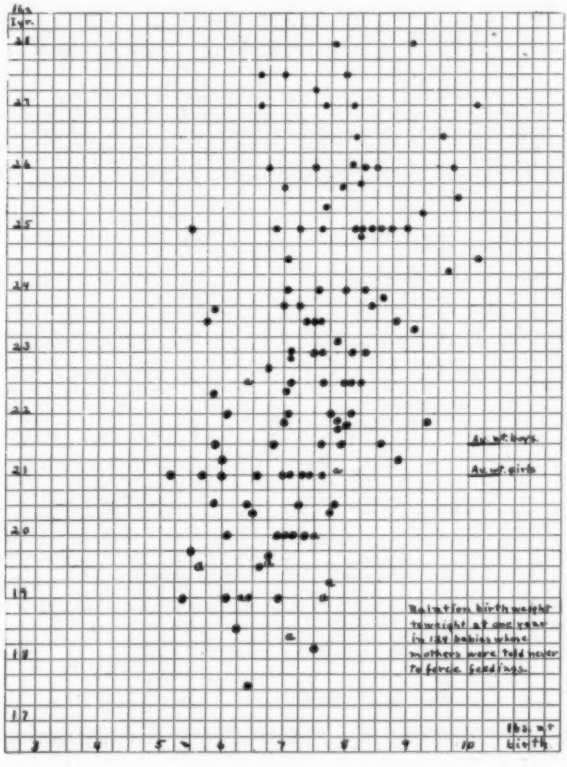

Fig. 1.

lack of cooperation rather than to the method advised. This failure is evidence of poor artistry on my part because if the physician's technic is good, obedience will follow.

Another scatter diagram (Fig. 2) is shown at this time because I think it demonstrates the fallacy of trying to feed babies on a caloric basis alone. This represents results on nearly 100 babies on the newly born service at the Evanston Hospital. On the ordinate are charted the number of ounces gained in the nine days between the beginning of lactation and discharge from the hospital. On the abscissa are found

the total number of ounces of food taken during this same period. Breast-fed babies are represented by circles, supplemented babies by dots. If there existed any close correlation between the number of calories taken and the weight gained, these dots and circles should arrange themselves close to a diagonal running from the lower left to the upper right part of the diagram. We find, on the other hand, a surprising scatter. This means that, while we know that there must be some relationship between the amount eaten and the weight gained, uncontrolled factors, even in these newly born babies, are so important that the caloric one is completely blotted out. The uncontrolled factors

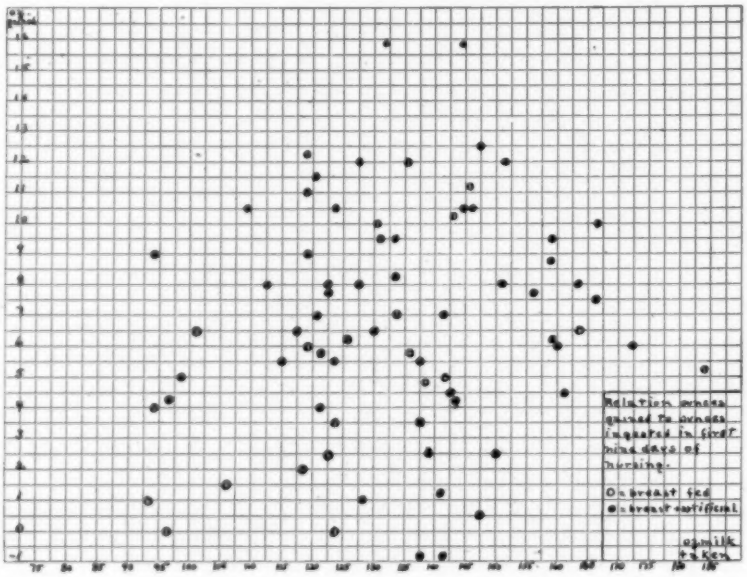

Fig. 2.

are, number and character of stools, vomiting, metabolism of the baby, his assimilation from the intestinal tract, urinary output, muscular activity, and body and room temperature. When a mother asks, "How much milk should my baby receive?" the answer should be, "Enough to make a good gain."

In the foregoing remarks, I have tried to illustrate a technic for feeding babies which will develop their appetite naturally, nourish them adequately, and result in an attitude toward food which will make available to our growing citizens any new discoveries in nutrition as they develop. In closing I should like to emphasize the fact that the really important consideration is not the actual technic which the individual physician uses, and not his exact prescription of diets, but his

attitude, his method of approach toward his patients. I appreciate that many men with a tolerant attitude toward children have obtained similar results in different ways. This is what makes child nurture an art, a medical as well as an educational or psychologic art. Success in art depends upon skill and knowledge of the trade plus the attitude toward its subject. Thus two equally skillful artists may interpret on canvas two entirely different personalities from the same sitter, and two equally talented musicians may from the same sheet of music, render effects which are dissimilar. In the same manner equally skillful physicians may, by their attitude toward their patients, produce entirely opposite results. The concept of attitude has a vague sound, but it is the most potent of ideas. If we could by a wave of the hand substitute in the minds of all people proper attitudes toward their fellows, we could with the next wave abolish war, police, armies, and most of the ills that ride civilization today.

Our attitude toward children must differ from that toward adults because the child is growing and the adult is static, jelled. We must respect the child more because we know so little about this growth of his and must depend so much upon it, and because in this unfolding, those of us who are optimistically inclined keep seeing ahead possibilities of greater perfection and charm than we actually find in our adult contemporaries. A respectful attitude toward growth will lead to thoughtful nourishment and care, much as a gardener tends his plants. It will never presume to model the child as a sculptor works his clay. The latter attitude would be clearly presumptuous in the present state of our ignorance about the intricate forces of growth and development which are so accurately working out the destinies of these small bodies. If the time comes when medicine can control stature and type, I hope to be able to retire gracefully and leave the field to those who take responsibility more lightly.

In approaching the child with the attitude of the gardener, we shall be of service in proportion to the skill with which we aid and direct natural growth. Pride in results will be justified by the extent to which we have applied the few known facts about the effect of environment (using the term in its broadest sense). In this conception our duty is to provide wholesome nourishment, adequate opportunity for growth, protection from disease, and to take care that factors interfering with normal growth are avoided. If abnormal tendencies appear, we must try to shape them into useful paths.

This idea that medicine and nutrition are able to control growth has put us in a very uncomfortable position. If we accept this position and pose on the high pedestal so created, we must be prepared to take the fall when the hard facts of truth knock out the props. If we deny

it, we lose caste in the public eye. Nevertheless, every argument of honesty and prudence bids us put both feet on solid ground, admit the facts, and prove our value in applying real scientific advances to the developing race. Firm in such a position, we shall find ourselves well fortified to withstand the brickbats of criticism which in these days fly about our ears. Seldom have men been armed with as brilliant an array of achievements as those given us by experimenters in the field of nutrition.

Another argument in favor of the adoption of such a tolerant attitude toward growth is found in the fact that this is exactly the attitude which has attracted the interest of the world to the other great professions interested in child welfare, the mental hygienists and the educators. "Respect the child's emotional life" has been the great cry of mental hygiene. "Respect and do not stunt the mental growth of your children" has been preached from the teacher's desk. If we are able to add to this the admonition, "Cultivate in each child his own physical perfection," the three great groups of workers can proceed in perfect harmony toward a goal which is reasonably attainable.

At the present time we are far from this accord. A well-known psychiatrist asserts that he has found forced feeding and its resulting difficulties to be the greatest single cause of behavior disturbances in his experience. I am assured by an experienced school-teacher that in her kindergarten the attitude of the children toward food is reflected in all their activities so that she can tell in a day's observation of any youngster whether or not his physician has insisted on quantity in food. In many instances our attitude toward children has alienated us from those whose assistance we should value most, allied workers in child welfare.

The technic here suggested for attaining our nutritional goals harmonizes with the ideals of such groups. It also adds the lure of the unknown to our future. We do not know what may be the heights of physical, mental, and emotional perfection which are inherent in human development, for children have always been blanketed by adult complacency. We, members of a great scientific fellowship, have never been complacent toward our task in eradicating and treating disease. We have always been striving for newer and better methods of attack, bending every energy for new truths, critical of our own results and questioning, always questioning what appears to be, to see if it really is. It is most consistent that in the questions of child development we keep this inquiring attitude. This, studiously applied toward the feeding inclinations of children, not only will bring us knowledge of natural trends but will probably be of therapeutic value in bettering their nutrition.

These remarks imply the need for such a tolerant attitude toward the child as a whole as well as toward his appetite. The likelihood that unprecedented unanimity of thought among the various professions interested in child welfare may thus come about is not the least of the benefits which might accrue. The child growing up today to meet the knotty problems of the near future will need all the knowledge he can get from the educators, all the emotional stability psychologists would provide, and all the physicial stamina we believe available through scientific nutrition. Would it not be an inspiring monument to the art of science if all forces interested could focus their activities harmoniously to give the youngsters their best possible opportunity to grow up? Such correlation is the task of tomorrow.

723 Elm Street.

A COMPARATIVE STUDY ON THE USE OF UNSWEETENED EVAPORATED MILK AND BOTTLED COW'S MILK IN INFANT FEEDING

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COW'S MILK in one form or another has been accepted by civilized countries as an infant food when mother's milk is not available. Since the chemical composition of cow's milk differs from mother's milk, various modifications of cow's milk for use in infant feeding have been tried in order to find a food that would most nearly approach mother's milk in digestibility. It has been found that boiled cow's milk is more easily digested than raw or pasteurized milk, that dried milk is more easily digested than boiled milk and that unsweetened evaporated milk is more easily digested than either boiled or dried milk (Marriott[1]), Brennemann[2]). Attempts have been made further to increase the digestibility of cow's milk by changing its buffer value, and by attenuating the curd. The reports on unsweetened evaporated milk feeding with addition of lactic acid (Marriott[1]) indicate that the buffer value is reduced so that the hydrogen-ion concentration during digestion approximates that of breast milk. Excellent results from the use of unsweetened evaporated milk have been demonstrated in a recent clinical experiment with 752 babies, comprising newly born, normal infants and sick babies (Marriott and Schoenthal[3]). Comparison was made with an almost equal number of babies on breast or on bottled milk formulas.

Equally enthusiastic results were reported by Brennemann[2] with unsweetened evaporated milk feeding in a group of sick babies. He obtained the same results claimed for unsweetened evaporated milk by Marriott. Brennemann tried unsweetened evaporated milk with and without lactic acid, alternately, and obtained the same satisfactory results with both the lactic acid and the sweet milk. Kerley[4] has been advocating the use of unsweetened evaporated milk for many years, claiming that it requires 50 per cent less digestive effort than does fresh cow's milk. Lowenburg,[5] in a clinical report on 175 babies, speaks most favorably of unsweetened evaporated milk.

Unsweetened evaporated milk is cow's milk from which approximately 60 per cent of the water has been removed by evaporation. It is then subjected to a process of homogenization, by means of which the fat particles are broken up and remain evenly dispersed throughout. The

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milk is then canned and sterilized by autoclaving at about 240 degrees F. It is subject to government inspection and control, as a result of which constancy of composition is insured to be not less than 7.8 per cent milk fat, nor less than 25.5 per cent total solids (Tobey[6]). Unsweetened evaporated milk is a safe food to use in infant feeding because of its freedom from bacterial contamination (Deming and Davis[7]).

Evaporation produces in milk certain changes which make it more easily assimilated. The process of homogenization in which the fat globules are broken up into smaller particles and evenly dispersed throughout the milk, prevents the formation of a cream layer. This homogenization, together with the heating, so acts upon the casein that during digestion it forms a small soft curd more nearly like that of human than any other milk (Rice[8]), (Brennemann[2]), (Marriott and Schoenthal[3]). In contrast to that of unsweetened evaporated milk, the curd in raw milk is large, firm and tough. The advantage of unsweetened evaporated milk in infant feeding is largely attributed to this difference in the size and character of the curd.

From the evidence in the literature (McCollum and Simmonds[9]), (Marriott and Schoenthal[3]), it appears that the vitamins, with the exception of vitamin C, are found in about the same proportion in unsweetened evaporated milk as in bottled cow's milk. Vitamin C, which is destroyed by heating, can readily be supplied by orange juice or tomato juice. Vitamins A and D, which are variable in quantity, can be supplemented by the use of cod liver oil.

CLINICAL REPORT

In spite of the favorable reports in the literature on the use of unsweetened evaporated milk in infant feeding, it was infrequently used in our feeding cases until December, 1930. In the clinical study we are recording, we alternated our artificially fed cases, as far as possible, between unsweetened evaporated milk and bottled cow's milk. No attempt was made to select cases. Our observations were carried on over a period of thirteen months, from December, 1930, to January, 1932. We have included in this report cases observed over a variable period of time, ranging from six weeks to one year. The study comprises 217 normal feeding cases, of which 110 were on unsweetened evaporated milk, and 107 on bottled cow's milk, also 13 premature infants, of which 8 were on unsweetened evaporated milk and 5 on bottled cow's milk.

I was assisted in this work by seven other physicians at the Los Angeles City Welfare Conferences. I saw about 50 per cent of the cases reported. At the onset of our clinical experiment, we were forced to combat the prejudice not only of a great many of the mothers, but of some

of the physicians as well. Because of their own prejudice against canned milk, several were reluctant to put the babies on this food. I am glad to report that before the experiment was over, the prejudice for the most part, was overcome, and the physicians were more cooperative in placing artificially fed babies on unsweetened evaporated milk.

Cooperation of the mother was solicited at the very start, and the importance of accurate information was stressed. In making our observations we were particularly anxious to note weight and height increases, the tendency to diarrhea, constipation or vomiting, the occurrence of rickets, and such developmental points as the time of the eruption of the first teeth, sitting, crawling, standing, walking.

The unsweetened evaporated milk formulas as used in our feeding cases, approximated the bottled cow's milk formulas. The greater concentration of unsweetened evaporated milk made it possible, however, for us to give a higher calorie content whenever it was found necessary. Estimation of the number of calories per pound was made on 43 of our feeding records in the unsweetened evaporated milk group and on an equal number in the bottled cow's milk group. The average was approximately 58 calories per pound, on unsweetened evaporated milk and approximately 53 calories per pound, on bottled cow's milk.

By far the greater number of babies were on modified milk formulas with Karo syrup added. In a smaller group, lactic acid or lemon juice was added to the formula. No appreciable difference was noted in our results with either acidified or nonacidified milk. Practically all babies were given orange juice or tomato juice, and cod liver oil before the third month.

For comparison of weight increases, we divided our cases into four groups. Some of the cases were included in more than one group, depending on the length of time observed. For example, a child started under observation at six weeks of age and observed until the end of one year was included in Groups I, II, III, and IV, since our average weights were obtained for three-month periods. No new cases over six months of age were added to our study. Group I includes all cases in which observations began before the child was three months of age. In this group, the average age at which observations began was six weeks and included 53 cases on unsweetened evaporated milk and 41 cases on bottled cow's milk. Group II includes all cases in which our observations began when the child was three months of age. In this group are 83 cases on unsweetened evaporated milk and 88 on bottled cow's milk. Group III includes all cases started at six months. In this group we included 64 on unsweetened evaporated milk and 67 on bottled cow's milk. Group IV includes cases from Group III in which observations were obtained for the age period of nine to twelve months.

Our observations on weight increases for the normal feeding cases at the end of each three-month period are shown in Table I.

TABLE I

NORMAL FEEDING CASES

		NO. CASES	AVERAGE GAIN PER WEEK	AVERAGE AGE STARTED	AVERAGE TIME OBSERVED	AVERAGE AGE OBSERVATIONS CONCLUDED
Group I	U. E. M.*	53	7.75 oz.	6 weeks	7 weeks	3 months
	B. C. M.†	41	6.62 oz.	6 weeks	7 weeks	3 months
Group II	U. E. M.	83	6.29 oz.	3 months	10 weeks	6 months
	B. C. M.	88	5.55 oz.	3 months	11 weeks	6 months
Group III	U. E. M.	64	4.47 oz.	6 months	12 weeks	9 months
	B. C. M.	67	3.80 oz.	6 months	12 weeks	9 months
Group IV	U. E. M.	24	2.79 oz.	9 months	11 weeks	12 months
	B. C. M.	30	3.04 oz.	9 months	11 weeks	12 months

*Unsweetened evaporated milk.
†Bottled cow's milk.

In addition to the above normal babies, we were able to observe a small number of premature infants: 8 were on unsweetened evaporated milk and 5 on bottled cow's milk. I am reporting, separately, one premature infant weighing 3 lb., 3 oz. at birth. This baby was placed on unsweetened evaporated milk immediately after birth, handled the food excellently, as evidenced by its average gain in weight of 8.5 oz. per week, for a period of nineteen weeks. Our observations on these premature infants are listed in Table II.

TABLE II

PREMATURES

	NO. CASES	AVERAGE GAIN PER WEEK	AVERAGE AGE STARTED	AVERAGE TIME OBSERVED	AVERAGE BIRTH WEIGHT
	7	4.19 oz.	10 weeks	18.85 weeks	4 lb., 4 oz.
U. E. M.	1	8.5 oz.	at birth	19 weeks	3 lb., 3 oz.
B. C. M.	5	3.94 oz.	13 weeks	26.80 weeks	4 lb., 3½ oz.

The height increases obtained on 86 infants during the first six months of life were as shown in Table III.

TABLE III

	NO. CASES	AVERAGE TIME OBSERVED	AVERAGE GAIN IN HEIGHT (PER MO.)
U. E. M.	43	3.70 months	1.06 inches
B. C. M.	43	3.58 months	1.04 inches

Other conditions noted were the frequency of vomiting, diarrhea, constipation, infections. These were reported to us as in Table IV.

TABLE IV

		AT ONSET ONLY	THROUGHOUT	OCCASIONALLY
Vomiting:	U. E. M.	14	2	11
	B. C. M.	3	1	10

		ONE ATTACK	TWO ATTACKS	MORE THAN TWO ATTACKS	TENDENCY PERSISTING THROUGHOUT OBSERVATIONS
Diarrhea:	U. E. M.	23	5	0	2
	B. C. M.	27	9	6	1

		AT ONSET	THROUGHOUT	TENDENCY
Constipation:	U. E. M.	2	13	9
	B. C. M.	6	9	11

		ONE COLD	TWO OR MORE COLDS	OTHER INFECTIONS, AS PYELITIS, OTITIS MEDIA, FURUNCULOSIS, ETC.
Infections:	U. E. M.	30	10	4
	B. C. M.	29	10	6

Observations were made on the occurrence of rickets. In addition to clinical observations, x-ray studies were made on 149 babies in this group, including 73 babies on unsweetened evaporated milk, and 76 babies on bottled cow's milk. One, two, and in some cases, three x-ray pictures were taken, depending on length of time observed, as shown in Table V.

TABLE V

	TOTAL NO. BABIES HAVING X-RAY PICTURES	NO. BABIES HAVING ONE X-RAY PICTURE	NO. BABIES HAVING TWO X-RAY PICTURES	NO. BABIES HAVING THREE X-RAY PICTURES
U. E. M.	73	31	36	6
B. C. M.	76	36	36	4

The x-ray pictures were taken at the Methodist Hospital and were all interpreted by Dr. Douglass R. MacColl. X-ray evidence of rickets were found in five cases on unsweetened evaporated milk and four cases on bottled cow's milk. The interpretation of the findings are tabulated in Tables VI and VII.

Developmental points, such as the time when the baby sat alone, crawled, stood alone, walked, and the eruption of the first teeth, were noted in the two groups in Table VIII.

TABLE VI

ROENTGENOLOGICAL FINDINGS OF RICKETS IN INFANTS ON UNSWEETENED EVAPORATED MILK

CASE	NO. OF PICTURES AND AGE WHEN TAKEN	NORMAL	ACTIVE RICKETS	DOUBTFUL ACTIVE RICKETS	HEALED RICKETS	NO ACTIVE RICKETS, POSSIBLY HEALED BEFORE FIRST X-RAY	FLARING ENDS OF DIAPHYSES	IRREGULARITY OF GROWTH	LINES OF INCREASED DENSITY
Case 1 (P. W.)	1st at 6 mo.					++	+		
	2nd at 11 mo.					++	+		
Case 2 (G. G.)	1st at 5 mo.	++							
	2nd at 11 mo.			+				+	
Case 3 (R. A.)	1st at 5 mo.			++			++		
	2nd at 10 mo.			++			+		
Case 4 (D. C.)	1st at 3½ mo.		++				++	++	
	2nd at 8½ mo.				++		++		++
	3rd at 13½ mo.				++		+		++
Case 5 (M. C.)	1st at 6 mo.		++				++	+++	
	2nd at 10 mo.				++		++		
	3rd at 15 mo.				++		+		++

+++ Marked. ++ Definite. + Slight.
Cases 1 and 2 did not show clinical evidence of rickets.
Cases 3, 4, and 5 showed few signs of clinical rickets.
Cases 4 and 5 did not receive cod liver oil or viosterol until after first picture was taken (although cod liver oil was ordered).

TABLE VII

ROENTGENOLOGIC FINDINGS OF RICKETS IN INFANTS ON BOTTLED COW'S MILK

CASE	NO. OF PICTURES AND AGE WHEN TAKEN	NORMAL	ACTIVE RICKETS	DOUBTFUL ACTIVE RICKETS	HEALED RICKETS	NO ACTIVE RICKETS, POSSIBLY HEALED BEFORE FIRST X-RAY	FLARING ENDS OF DIAPHYSES	IRREGULARITY OF GROWTH	LINES OF INCREASED DENSITY
Case 6 (J. E.)	1st at 5 mo.		+				+	++	
Case 7 (Wm. S.)	1st at 3½ mo.	++							
	2nd at 7½ mo.			++				+	
	3rd at 11 mo.				++				+
Case 8 (L. P.)	1st at 5 mo.					+			+
	2nd at 8 mo.					++			++
Case 9 (A. C.)	1st at 5½ mo.					++			++
	2nd at 9 mo.					++			++

+++ Marked. ++ Definite. + Slight.
Case 6 did not attend regularly and our records are incomplete.
Cases 6 and 8 very doubtful if cod liver oil was given (although ordered).
Cases 7 and 9 showed no clinical evidence of rickets.
Case 8 showed clinical evidence of rickets.

TABLE VIII

		NO. CASES	AVERAGE AGE
Sat alone	U. E. M.	59	26 weeks
	B. C. M.	52	26 weeks
Crawled	U. E. M.	41	34 weeks
	B. C. M.	39	35 weeks
Stood alone	U. E. M.	32	42 weeks
	B. C. M.	29	41 weeks
Walked	U. E. M.	23	49 weeks
	B. C. M.	25	48 weeks
Eruption first tooth	U. E. M.	51	30 weeks
	B. C. M.	52	32 weeks

SUMMARY

From the data obtained on 217 normal feeding cases and 13 premature infants, we draw the following conclusions:

1. The average normal baby is able to assimilate unsweetened evaporated milk as well as or better than bottled cow's milk (pasteurized or boiled) during the early months of life when milk constitutes the sole food, as evidenced by their weight increases.

2. Similar good results were obtained in the premature infants.

3. The gain in height is practically the same on both foods.

4. Vomiting at the onset of the feeding experiment was reported more frequently in the unsweetened evaporated milk group. Wherever the mother could be persuaded to continue on the unsweetened evaporated milk, especially if a good gain in weight was evidenced, the vomiting in most cases was subsequently reported as "just spitting up."

5. Diarrhea was not as frequent on unsweetened evaporated milk as on bottled cow's milk.

6. Constipation although frequently complained of, was not more marked than on bottled cow's milk.

7. The relative frequency of infections as evidenced by colds, etc., was about the same in both groups.

8. The relative incidence of rickets was a little more marked in the unsweetened evaporated milk group. However, that was undoubtedly influenced by the fact that the babies showing active rickets did not receive cod liver oil or viosterol until the rickets was discovered by roentgenogram—this in spite of the fact that cod liver oil is included routinely in our feeding régime.

9. The developmental points as sitting alone, crawling, standing, walking, and eruption of the first teeth were almost identical for both groups.

CONCLUSION

1. From our own observations on weight increases, we conclude that in the early months of the infant's life, unsweetened evaporated milk has certain advantages in infant feeding. In every other respect, there

seems to be no appreciable difference in unsweetend evaporated milk and bottled cow's milk.

2. From the standpoint of economy, availability, sterility, uniformity of composition, easy digestibility, unsweetened evaporated milk has decided advantages over bottled cow's milk in infant feeding.

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116 TEMPLE STREET.

DIFFUSE SCLEROSIS AND MALFORMATIONS OF THE BRAIN IN A CHILD TWO YEARS OLD

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AND

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THE hereditary and congenital factors in the infantile and juvenile groups of diffuse sclerosis have attracted considerable attention in the last twenty years. These considerations are based chiefly upon clinical observations and not upon pathologicoanatomic findings, since the disease has been observed in several members of the same family in infancy and childhood. The groups in question have been classified by Bielschowsky and Henneberg[1] as Leucodistrophia progressiva hereditaria within which three subdivisions can be made: (1) Peliczaus-Merzbacher's disease; (2) type Krabbe; (3) type Scholz. The importance of heredity in Peliczaus-Merzbacher's disease is beyond doubt as the disease has been followed through three generations. In the other two the hereditary factors are probable but are not yet sufficiently investigated. In none of the observations so far reported have malformations of the body or the brain been found.

In the following we wish to present a case in which an unusual combination of malformation and diffuse sclerosis occurred.

Clinical Report.—D. M., two years old, white male, entered the pediatric clinic April 18, 1931, because of retarded mental and physical development. He was a full-term, nine-pound baby with polydactylism and syndactylism of both hands and feet. No other abnormalities were observed. He was breast fed for the first year, was given orange juice and cod liver oil at the proper time and cereals and vegetables during the latter part of the year. Some time during his first three weeks he began to vomit. This has continued more or less continually to the present time, and has become projectile in character. The bowel movements have been normal. He had been treated for pyloric stenosis but no evidence of a gastrointestinal lesion or definite abnormality responsible for the vomiting could be demonstrated. The vomiting usually occurred soon after a feeding. The teeth began to erupt at the eighth month. He held his head up and sat up at ten months. He has never talked or walked.

There is a history of measles and of frequent upper respiratory infections. Soon after his first year the deformities of the hands and feet were corrected. There is a history of a fall on a cement pavement from a height of about five feet at the eighteenth month but no details can be secured.

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Present Illness.—There have been marked evidences of retrogression both mentally and physically during the second year. A relationship to the fall has been suggested. Since this time he has been unable to support his head or sit up. There have been no convulsions. He has gradually become spastic in all his extremities.

Physical Examination.—Weight 19 pounds, height 32 inches, temperature 99.4° F. The patient is somewhat undernourished and underdeveloped, and is unable to hold his head or sit up. The skin is pale. The panniculus is thin except over the abdomen where it is moderate in amount. There is a general slight adenopathy. The tendon reflexes are variable and difficult to elicit, at times hyperactive. No Babinski, Oppenheim or Kernig can be demonstrated. The pupils are round, equal, react to light and in accommodation. Ophthalmoscopic examination shows a well-marked secondary optic atrophy. The fontanelles are closed. The hair is fine.

Fig. 1.—Ventriculogram. Left lateral view. Ventricular outlines faintly retouched. Anterior and posterior trephines, round defects in left skull showing through from right. Lateral ventricle well filled, third ventricle beneath, foramen of Monroe between. At vertex collection of subdural air faintly outlined.

The head is of the hydrocephalic type. The infant appears mentally deficient, grimacing and yawning much of the time. The thyroid and joints are negative. The muscles are somewhat wasted. The extremities are definitely spastic. Supernumerary digits and webbing of the hands and feet have been cared for surgically.

Laboratory Findings.—Urin negative. Blood, 5/2/31: hemoglobin 80 per cent (Sahli); red cells 4,290,000; white cells 4,900; polys 51 per cent; eosinophiles 0; basophiles 1 per cent; small lymphocytes 40 per cent; monocytes 8 per cent; platelets normal; no abnormal red cells. Blood, 5/5/31: hemoglobin 70 per cent, white cells 9,100. Blood Kahn negative.

Progress Notes.—May 3, 1931. Third day in hospital. Developed a temperature of 105.6°. During the night of the third day he became restless, cried a great deal and sweat profusely. His appetite failed. In the afternoon he became rigid, the eyes staring. He was unable to swallow. When food was forced he vomited it immediately.

May 4, 1931. On the night of the fourth day the extremities became stiff. In the morning he was hungry. He continued to perspire freely. As the day advanced he became more comfortable.

May 5, 1931. Fifth day. Temperature reached 100°. Leucocytes 9,100.

May 6, 1931. Sixth day. Temperature reached normal. It was 103° eight hours after operation. A ventriculogram was made. An incision one inch long was made about three-quarters of an inch from the midline, one and one-half inches above the orbit on the right side. The skull was perforated with a Hudson drill. The dura was opened and an enormous amount of fluid was found in the subarachnoid space. A vertical incision was then made in the occipital region about one and one-half inches from the midline. The skull was opened again. The subarachnoid space was the same as before. A needle was inserted and a large

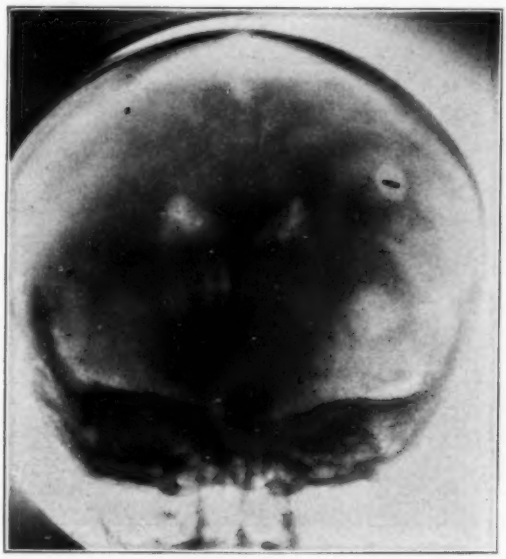

Fig. 2.—Ventriculogram. Anteroposterior view. Trephine openings on right. Anterior horns show normal contour, some dilatation, no midline shifting. Portion of ventricle bodies well known. Moderate amount of subdural air at vertex sharply defined.

amount of fluid escaped. The brain seemed to drop away from the dura. About 200 c.c. of fluid were withdrawn, a like amount of air injected, and the wound closed. (Dr. Max Peet.)

X-ray of the head following this procedure showed satisfactory filling of the entire ventricular system (Figs. 1 and 2). There was moderate symmetrical enlargement of the posterior horn of both ventricles. The films in the supine position suggested mild dilatation of the anterior horn. The foramen of Monroe, third ventricle, aqueduct of Sylvius, fourth ventricle, and the basilar cistern appeared fairly well defined and showed no gross abnormality. A striking feature of the examination was the gross area of aeration external to the cortex, without any delineation of the convolutional markings, strongly suggestive of air in the subdural space. The air shifted with change in position. (Dr. F. Hodges.)

DATE	TEMP.	PULSE	RESP.	
May 7, 1931	105	140	30	Took liquids well. Rigid in afternoon.
May 8, 1931	103	140	30	Comfortable. Urine negative.
May 9, 1931	101	140	32	Slept a great deal. Nauseated.
May 10, 1931	101	130	30	Comfortable.
May 11, 1931	101	140	30	Comfortable.
May 12, 1931	102	120	22	Free discharge from left ear. Irrigations.
May 13, 1931	104	140	40	Discharge from ear greater. Irrigations. Weaker.
May 14, 1931	103	160	35	Marked discharge from ear. Died 7:20 P.M.

Clinical Diagnosis.—Malformation of the brain, type undetermined.

Pathologic Report.—Postmortem examination revealed a peculiar brownish color of the skeletal musculature, an open foramen ovale, syndactylism and acute otitis media.

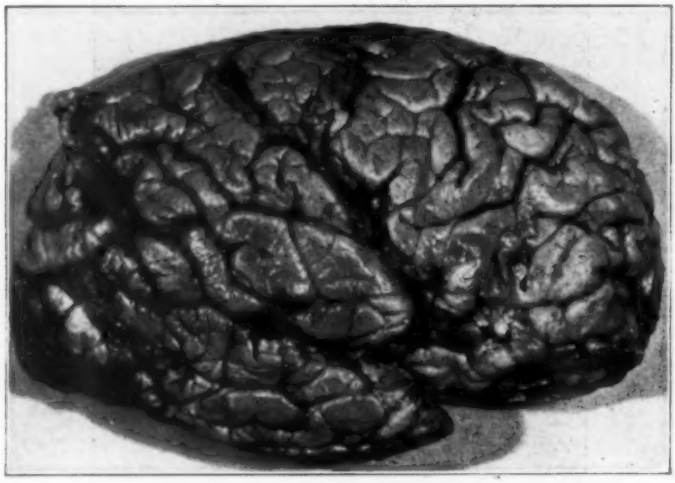

Fig. 3.—Cerebral sulcus short and wide joining the Sylvian fissure. Central convolutions very plump. Temporal lobe triangular in shape. Photograph.

The brain appeared to be large and of a peculiar rubber-like consistency. Both frontal lobes were well developed and their convolutions were of normal type but very plump. In the parietal lobes the central sulcus showed an abnormal course, being short and wide and joining the Sylvian fissure. The main branch of the latter was abnormally large and was directed sharply upward.

The temporal lobes were approximately triangular in shape and almost completely divided by a very deep superior temporal sulcus (Fig. 3). The convolutions of the occipital lobes were without striking deviations from the normal, but those of both median surfaces were quite irregularly shaped. The sulcus cinguli was rather short and its median part divided into many small branches. The sulcus parieto-occipitalis and the calcerine were, however, approximately normal in shape.

At the base of the brain the convolutions appeared primitive but the resulting picture was a fairly normal one.

Histologic Examination.—The leptomeninges show no abnormalities.

Cortex.—The cytoarchitecture is everywhere of a six layer type and the parenchyma is severely affected. There is a distinct reduction in number of cells, so that the layers cannot be clearly outlined and at places an irregular cell arrangement is present.

Fig. 4.—Complete destruction of myelin in cortex. Diffuse demyelinization of the white matter and the basal ganglia. U-fibers better preserved. Photograph. Weigert stain. (½ natural size.)

Fig. 5.—Myeloarchitecture destroyed; U-fibers visible; numerous perivascular foci in the white matter. Weigert stain. Photograph. (½ natural size.)

The still preserved elements are greatly changed, shrunken, void of tigroid, the nuclei stain rather intensively in some areas. Many cells are reduced to shadows but in all instances the change is an unspecific one. There is no glial activity in the cortex, no vascular proliferation, no inflammatory change of any kind.

Bielschowsky preparations disclose an almost complete absence of axis cylinders throughout the cortex and in accord with this, Weigert sections show an almost complete destruction of the myeloarchitecture. Here and there a few weakly stained myelin radii can still be traced (Figs. 4, 5, 6).

With the thionin stain the white matter assumes a pinkish-blue color in which small linear foci, running in all directions, stain somewhat more deeply. These are visible microscopically.

Throughout the white matter there are numerous plasmatic astrocytes, uniformly distributed (Fig. 7). They form dense rings in the perivascular spaces in which they are frequently mingled with gitter cells. These peculiar cell accumulations are responsible for the above-mentioned, grossly visible foci.

There is a moderate amount of gitter cells in the white matter also, but they contain surprisingly little phagocytosed lipoids. In the blood vessel walls a few

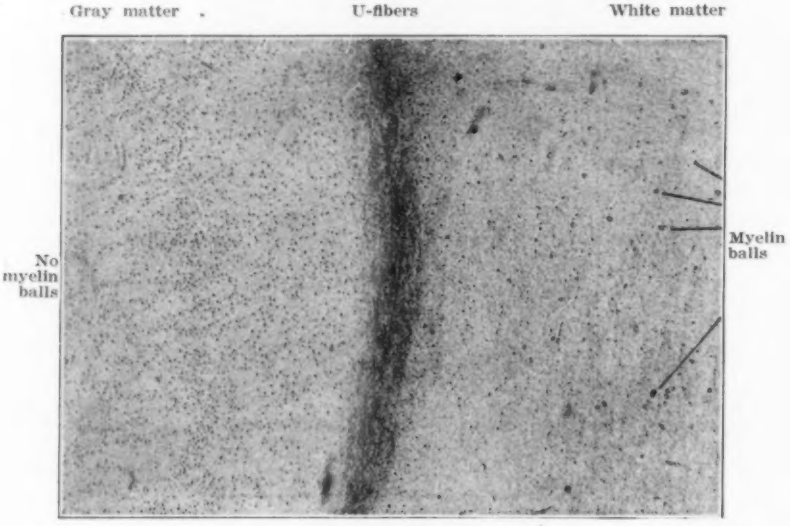

Fig. 6.—Myeloarchitecture destroyed. U-fibers visible; white matter almost completely demyelinated, contains numerous myelin remnants. Weigert stain. Photomicrograph. Zeiss Planar 20 mm. Ocul. 1.

fat droplets may be seen. In some areas, particularly in the frontal and temporal lobes the gitter cells increase in number and are studded with brownish pigment.

The study of sections stained by Cajal's gold and silver and Hortegas silver carbonate methods failed to reveal additional details. In the white matter of the central region there is a focus of incomplete necrosis the size of a fifty-cent piece, in which but few nuclei can be traced.

Weigert preparations reveal the true extent and character of the disease: a grayish black impregnation of the myelin can be obtained and throughout the entire white matter there are numerous yellowish foci and lines which do not take up the stain at all (Figs. 4 and 5). In the center of these lesions a blood vessel can frequently be seen. These foci are identical with those described in Nissl sections. The diffuse grayish-black tinge of the white matter is somewhat accentuated by a deeper impregnation of the U-fibers, which are therefore more distinctly outlined (Figs. 4, 5, and 6).

The myelin disintegration is unusually far advanced and evenly distributed. The outstanding feature of this phenomenon lies in its ubiquity; the conditions are fundamentally different from those usually seen in diffuse sclerosis in which the

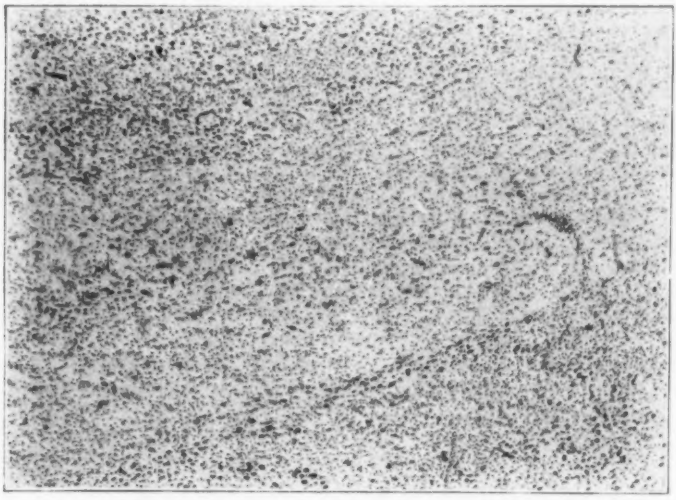

Fig. 7.—Astrocyte proliferation in the white matter (pons). Nissl stain. Photomicrograph. Zeiss Planar 20 mm. Ocul. 1. Small bodies, astrocytes.

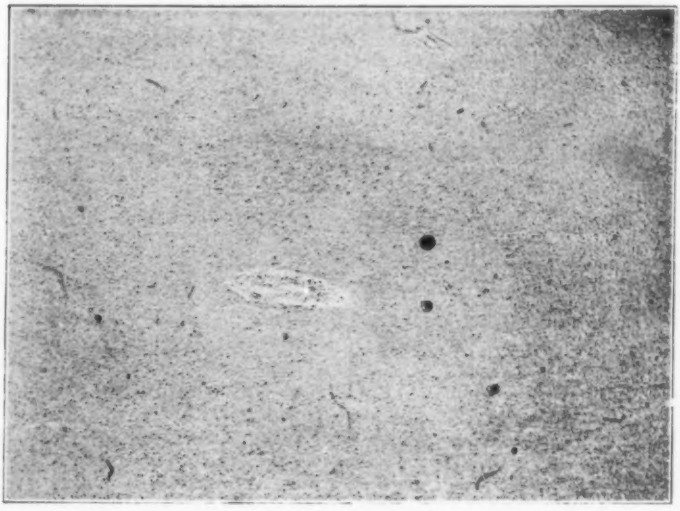

Fig. 8.—Perivascular lesion with complete demyelination. White matter is better preserved in the right side of the picture. Blood vessel in the center of the lesion. Weigert stain. Photomicrograph. Zeiss Planar 20 mm. Ocul. 1.

upper layers of the white matter are frequently better preserved than the deeper, the transition being a more gradual one. On the contrary, in the case in question

no transitional phases are present and the whole white matter has been affected with a remarkable evenness, the U-fibers being the only exception (Figs. 4, 5, and 6).

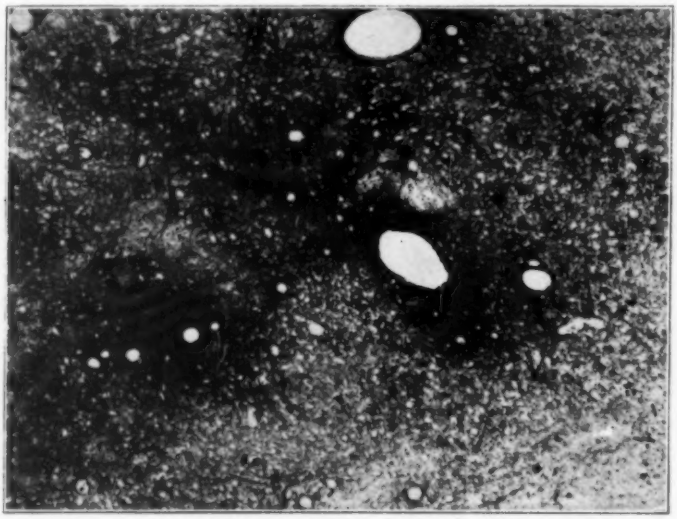

Fig. 9.—Perivascular glial rings, dense polymorph network throughout the white matter. Holzer-stain. Photomicrograph. Zeiss Planar 20 mm. Ocul. 1.

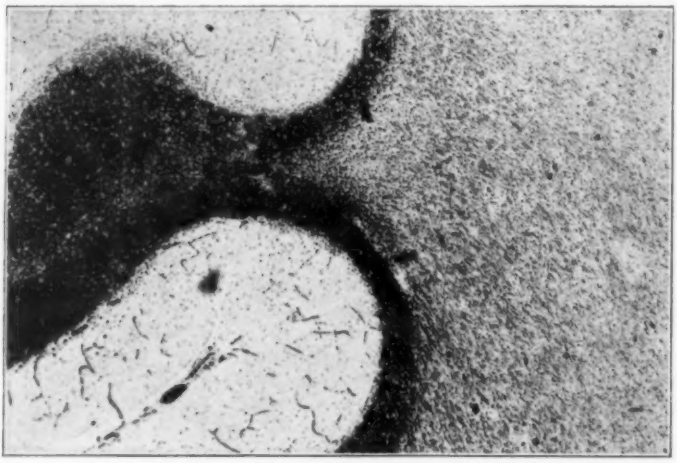

Fig. 10.—Advanced demyelination of the white matter of the cerebellum. Weigert stain. Photomicrograph. Zeiss Planar 20 mm. Ocul. 1.

More detailed histologic analysis reveals three types of degenerated myelin sheaths: (1) Very fine, slightly tortuous ones, staining but weakly. They are present in considerable numbers. (2) Considerably larger sheaths, edematous and

uneven in width, also weakly stained and present in moderate numbers, and (3) myelin disks or balls, remains of completely destroyed myelin structures, scattered everywhere.

In the region of the perivascular foci hardly a single sheath remains so that the myelin disintegration here must be considered complete (Fig. 8).

The corpus collosum and the capsula externa and interna show the same grayish-black tinge as the white matter in general.

In the white matter, Bielschowsky sections reveal a correspondingly advanced reduction of axis cylinders, the ones remaining being very fine, tortuous, and uneven.

Holzer's glia fiber stain brings out an extremely dense polymorph fiber production which occupies the whole white matter, nowhere, however, invading the gray. The fiber production is particularly dense around the blood vessels, giving rise to pictures corresponding to those in Nissl and Weigert preparations (Fig. 9).

The basal ganglia are also involved in the disease. The putamen, caudatum and thalamus are distinctly outlined in Nissl sections, but the parenchymatous elements are greatly reduced and stain poorly. This type of degeneration corresponds to that of the cortex in every respect. The glia forms astrocytes and gitter cells in moderate amounts without tendency to perivascular accumulations.

The condition of the pallidum is a different one: the parenchyma is here destroyed and but few glial nuclei are present. It is a condition of incomplete necrosis as described in the central region of the white matter.

In all these formations there is a moderate polymorph gliosis present.

The demyelinization of the basal ganglia is also far advanced. The myelin is somewhat better preserved in the putamen and the caudate nucleus but the examination under high power reveals everywhere the same findings as in the white matter.

In the pallidum and the thalamus there is a most severe demyelinization. The histologic pictures in the subthalamic nuclei, pons, medulla and cerebellum (Fig. 10) are the same as in the white and the gray matter of the brain: diffuse proliferation of astrocytes and gitter cells, pronounced but unspecific degeneration of parenchyma, polymorph gliosis and advanced demyelinization.

Both nerves are completely demyelinated.

Inflammatory reactions and connective tissue response are nowhere present.

The disease is symmetrical.

The spinal cord is not available for study.

CLINICAL SUMMARY

A two-year-old white male, born of normal and healthy parents, showed signs of malformations of hands and feet. The child suffered from an intestinal ailment the first month of life and its mental development was retarded. During the first year there was some mental development and the child could hold its head erect and learned to sit. It lost these abilities in the second year, during which an increasing spasticity of extremities and body, associated with bilateral optic atrophy, were noted. According to the clinical record there were no pathologic reflexes, the spasticity being the dominating symptom.

PATHOLOGICO-ANATOMICAL SUMMARY

There was a puzzling combination of malformations of the hands, feet, heart, and brain. The convolutions were abnormally shaped.

The consistency of the brain was rubber-like and on frontal section there were numerous small white foci in the white matter.

The histology is characterized by unspecific degeneration of parenchyma, a universal destruction of myelin in both gray and white matter associated with a moderate glia cell response, with perivascular accentuation and a universal polymorph glia fiber production. It represents a degenerative type of brain disease.

COMMENT

For the classification of this case the demyelinization of the brain seems to be the dominating factor and calls our attention to the group of diseases known as "diffuse selerosis." A cautious comparison, however, shows that this case is not identical with those so far reported as belonging to that group.

By comparison, our case has congenital stigmata such as malformations of body and brain which have not so far been observed in connection with diffuse sclerosis. The significance of congenital and hereditary factors in some groups of diffuse sclerosis has been frequently emphasized and discussed. They have been established, however, for only a single family reported by Peliczaus-Merzbacher. No definite genetic data have been given in the few sporadic cases reported obviously representing the same condition. Even more unsatisfactory is the knowledge of hereditary conditions in the observations of Krabbe, who described diffuse sclerosis in siblings during the first year of life. Finally, Scholz reported the same disease in two brothers, eight and nine years old respectively, who had been healthy before the onset. Several members of the family showed nervous disease, but the connection is not quite clear.

The few observations so far published and the great difficulties which genetical investigation encounters make it difficult to decide whether there really was no heredity factor in those cases in which it was not reported or whether such factors were not brought to the attention of the examiner. Our own case illustrates this very well; in spite of the multiple malformations described, the parents knew nothing of similar disease in the family, a statement which is not very convincing in view of the fact that syndactylism is almost always a dominant trait.

It is further difficult to decide whether there are clinical trends similar to those seen in leucodystrophia hereditaria as the clinical observation in our case was very short and the child entered the hospital but a few weeks before death.

The only point of accord between our case and those of Merzbacher is that the disease began very soon after birth. In Merzbacher's observations a horizontal nystagmus appeared after three months.

Half a year later weakness, ataxia, paresis, and contractures of the lower extremities were noted. In spite of this the patients learned to walk a little at the age of three. But speech and gait were frequently lost toward the end of the third year. Later on cyanosis and atrophy of the lower extremities were observed. Feeble-mindedness and idiocy were frequent. The course of the disease was an exquisite, chronic one, lasting for two or three decades, the oldest patient being fifty-two years old at the time of death.

The patients of Krabbe were infants or young children, the clinical course was stormy and the disease was terminated by death a few days or weeks after the onset. The heredity was not discussed by the author. Finally Scholz described diffuse sclerosis in two siblings toward the end of the first decade, who had been healthy before the onset of the disease. The duration of the disease was several years.

Pathologicoanatomically comparison is possible to some extent, though combined malformation of body and brain in diffuse sclerosis has not been previously observed. Moreover there are important histologic deviations: the demyelination is equally developed and distributed in cortex and white matter, basal ganglia and brain stem, the U-fibers being the only exception. Diffuse sclerosis in general and one of its subdivisions, the so-called Schilder's disease, do not show such findings. An advanced demyelination of the cortex in Schilder's disease has been reported by Jakob, but it involved only certain circumscribed areas and not the whole brain as in our case.

The myelin destruction in the white matter differs greatly in Schilder's disease, in which there are one or a few large foci in the white matter, the superficial layers of which are better preserved. On the contrary in our case no focal lesions have been seen, the destruction of the myelin being equally distributed throughout the entire white matter. This explains also the extent and the type of the gliosis.

In accord with the latter the glial cell response shows the same mode of distribution so that with the exception of the perivascular glial rings no foci could be observed.

The histologic findings seem to indicate that the disease had already reached its terminal stage; the far advanced destruction of the myelin, the extent of the glia fiber production, the negligible amount of lipoids and the countless myelin balls make such an assumption reasonable. Because of this it is very difficult to reconstruct the earlier stages of the condition. Two possibilities have to be considered: first, that destruction of the myelin did not take place because of retardation of myelin development. In other words we are dealing with a myelin aplasia in a malformed brain. Secondly, myelin development occurred but had undergone destruction in its nascent state.

Merzbacher advanced the former view in discussing his case but subsequent investigators have disagreed with him (Spielmeyer).[6] In our case there are no histologic reasons to assume an aplasia, on the contrary the findings seem to indicate that myelin formation had taken place as there are numerous degenerated myelin sheaths and countless remains of its disintegration. This disintegration is equally distributed and obviously synchronous in all parts of the nervous system, so that no circumscribed foci could result.

For the interpretation of this peculiar picture the perivascular gliosis seems to offer certain clues. It is just as universally distributed as the demyelinization and so powerfully developed that it can be seen grossly. All this suggests that the blood vessel system was involved in the disease very early and intensely. Most likely the perivascular myelin was destroyed first as none is left in the glial belts.

The nature of the responsible agent cannot be determined because of lack of definite histologic changes in the blood vessel walls proper. We are probably dealing with a congenital anomaly of the meningovascular barrier in a malformed brain. Such a generalized blood vessel change would expose the myelin to the influence of pathologic metabolic products and bring about the described picture.

For the reasons stated we must be cautious in classifying this case. The groups so far adopted are based upon so few observations that every new case tends to change our conception of them; on the other hand it is not permissible to establish a new subdivision represented by a single case. This case is therefore tentatively added to the groups of "diffuse sclerosis" on the basis of the demyelination, an important feature manifested in common by all the cases previously reported.

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THE TOXEMIA OF UPPER INTESTINAL OBSTRUCTION, ITS NATURE AND TREATMENT

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OBSTRUCTION of the upper bowel, aside from tumor masses and postoperative factors, occurs with a frequency of over 80 per cent during infancy and childhood. Cope[1] reports that 70 per cent of the cases of intussusception appear at two years of age or less. Pyloric stenosis, relaxation of the mesentery, adhesive bands, strangulated hernia, and foreign bodies are other common causes of obstruction at this period. The resulting inhibition of normal intestinal motility is attended by grave consequences. The higher the level of the lesion in the gastrointestinal tract, the more rapid and severe the onset of the intoxication. A fatal termination results when the cause of the obstruction is not removed. In a great many cases it is comparatively easy to relieve the obstruction, but the discouraging factor has been that death is likely to occur.

In 1908 Scudder[2] reviewed a large series of cases of intestinal obstruction. He stated that the treatment was purely surgical relief of the existing obstruction, the mortality being considerably over 50 per cent. In 1920 Richardson[3] presented another large series of intestinal obstruction. By comparison he concluded from this study that surgical procedures and experimental evidence available at that time offered no improvement in the treatment of these patients over that reported in Scudder's series, presented twelve years earlier. His plea was for earlier diagnosis and early surgical intervention, before the onset of the ensuing toxemia which occurred with great rapidity, death occurring in his series where the symptoms of obstruction had been of two and six-tenths days' duration. He concluded that the institution of surgical measures after the onset of toxemia was a worthless procedure. Jefferson Miller[4] ventures to say that 20 per cent of these patients are operated when there is not more than a 1 per cent or 2 per cent chance for their recovery. His analysis of 343 surgical cases of intestinal obstruction shows a mortality of over 60 per cent.

The death which occurs from intestinal obstruction has long been believed to be a result of the late severe manifestations of the toxic state accompanying the obstruction. Although many theories have been advanced as to the nature of the toxemia, exhaustive experi-

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mental and clinical investigations have, until only recently, failed to reveal its nature. The toxemia was considered by Murphy and Vincent[5] to be of bacterial origin, arising from bacteria in the proximal obstructed gut. Whipple[6] suggested the presence of a proteose above the obstruction as a causative factor. Murphy and Brooks[7] believed the consequent interference with the circulation to be the essential factor in allowing abnormal absorption of the intestinal contents. Constipation, starvation, and dehydration have likewise been presented as the cause of this complex.

Formerly, a patient with intestinal obstruction, dehydrated from vomiting, with florid complexion, and dry skin was thought to have a severe acidosis, due to mechanical interference of the intestinal tract. Advancement in knowledge of the blood chemistry and its application, experimentally and clinically, now discloses that alkalosis intervenes instead of acidosis. Recent investigations show that the toxemia has definite characteristics to guide in the diagnosis and treatment of such a patient. At the present time it is recognized that, with a case of acute intestinal obstruction in which surgical intervention is necessary for relief, preliminary medical treatment is of utmost importance. Proper medical treatment for the toxemia combined with surgery will beyond question lead to a decided lowering of the mortality rate.

REPORT OF CASE

The following case is presented to demonstrate the value of combined medical and surgical treatment.

J. S., boy, aged seven. Was seen on third day of illness. Three days previous he had suddenly begun to vomit after his evening meal. During the intervening period all foods and fluids taken had been promptly vomited. During the first evening's illness one normal stool had been evacuated. On the second day small, mucous, starvation stools containing flakes of blood with no fecal material had been passed. At the onset of the illness the child had agonizing abdominal pains accompanied by rigidity of the entire abdomen, which led the attending physician to consider appendicitis as an etiologic factor. The temperature, however, was not elevated, the leucocyte and differential counts were normal. The abdominal pain and rigidity would at times disappear only to recur at a later period. The vomiting persisted during the second and third days and became bile-stained.

At examination was noted a well-developed boy, presenting an extremely toxic appearance, the face carrying an anxious expression and eyes appeared to be protruding. The skin was extremely dry and inelastic. Respiration was normal. The breath sounds were also normal. The heart tones were good, the rhythm accelerated. He appeared to be having severe abdominal pain, remaining in bed with thighs flexed. The abdomen was sunken and general tenderness was present over its entirety. The spleen and liver were not enlarged. No tumor masses could be palpated. The leucocyte count was 12,374 with 71 per cent polys. Rectal examination was negative. The child had voided but scanty amounts since the onset of the illness. Catheterization at the time of examination revealed the bladder to be empty.

The condition was presumed one of obstruction of the upper bowel, the vomitus being bile-stained but containing no fecal matter. A chemical analysis of the blood, shown below, was confirmative of the diagnosis of upper intestinal obstruction.

The sodium chloride was found to be depleted, 330 mg. per 100 c.c. Nitrogen retention was present, urea nitrogen 89.4 mg. per 100 c.c. The carbon dioxide combining power of the plasma was elevated, 118 volume per cent. Because the patient seemed to be in a moribund condition, 400 c.c. of 2 per cent sodium chloride and 10 per cent dextrose solution were given intravenously. After four hours, examination of the blood chemistry showed the plasma chlorides at 475 mg. per 100 c.c.; urea nitrogen 41.2; carbon dioxide combining power 65 volume per cent. With these approximately normal levels a laparotomy was now considered advisable.

After lavaging the stomach, an incision was made, meeting an obstruction due to an adhesive band 8/10 cm. in width between the duodenum and gall bladder, which completely encircled the gut. The band was severed and a gastroenterostomy performed, no effort being made to evacuate the bowel content above the obstruction. Immediately following the operation 500 c.c. of 2 per cent sodium chloride and 10 per cent dextrose were again given by hypodermoclysis. The following day plasma chlorides were at 465 mg.; urea nitrogen 39; carbon dioxide combining power 70 volume per cent.

Although the child seemed to be recovering and had retained small amounts of fluids by mouth, only scanty proportions of urine had been voided. Consequently, glucose, for the diuretic effect, was indicated and, because the plasma chlorides were at 465 mg., only 1 per cent salt solution containing 10 per cent dextrose was given, 300 c.c. intravenously. On the second day after the operation clinical symptoms had considerably lessened. The patient was voiding. Chemical analysis of the blood revealed normal values for the chlorides, urea nitrogen and carbon dioxide combining power: chlorides 495 mg., urea nitrogen 37 mg., carbon dioxide combining power of plasma 64 volume per cent. The convalescence was uneventful.

In 1919 Tucker, working at The Mayo Clinic, first recognized the similarity between the toxic syndrome of intestinal obstruction and uremia. His investigations revealed that nitrogen retention was present in eight cases of obstruction following gastroenterostomy. Clinical contributions were subsequently added by Dixon,[8] McVicar[9] and their associates at The Mayo Clinic. Observant of these clinical reports, Whipple[10] first carried out blood chemistry investigations in dogs in which intestinal obstruction had been produced. Recently, Haden and Orr[11] have presented illuminating investigative studies on the effect of experimental obstruction, while Walters and Bollman[12] have called attention to comparable disturbances in the blood chemistry in association with duodenal fistula.

The outstanding laboratory findings which have been observed during these various investigations can be summarized as:

1. Nonprotein nitrogen retention, as indicated by the high blood urea.

2. Increased alkalinity of the blood, as shown by the increased carbon dioxide combining power of the blood plasma.

3. Hypochloremia, marked by a fall in the blood chlorides.

The nitrogen retention suggests nephritis, where there is also urea retention. In renal insufficiency, however, the disturbance in the acid base equilibrium is toward acidosis, and chloride retention is present instead of depletion. Haden and Orr[13] believe that the rise in the nonprotein nitrogen is due to two factors: (1) The concentration of the blood plasma, from the great fluid loss, and (2) from the great increase in the tissue destruction taking place. The protein destruction may result from dehydration of the tissue cells or from a circulating toxin. Dragstedt, et al.,[14] have shown that the toxic element in obstructing fluids causes a marked increase in gastric and intestinal secretions, hence a greater chloride loss results from the increased secretion into the stomach and intestines, as well as from vomiting. Rowntree[15] from data collected from various sources illustrates that secretion of digestive fluids taken together may total five to seven liters daily, in adults. This is two to three times the volume of blood plasma that is the immediate source of these secretions. In toxemia there is a greater loss of chloride than sodium. The excess sodium combines with carbonic acid to form sodium bicarbonate, hence causing an alkalosis, which is measured by the carbon dioxide combining power of the plasma.

Dehydration is a constant finding in upper intestinal obstruction. This can be determined by hematokrit readings, which show an increase in the percentage of the red blood cells, and by the determination of the protein content of the plasma. A marked increase of the blood fibrin is present, which is much accelerated in any condition where there is tissue injury. Haden and Orr have demonstrated that other changes take place which may be important factors in the development of symptoms and death. The blood viscosity[16] is increased, thus impairing the circulation and interfering with oxygenation of the blood. The sedimentation[17] test shows a marked decrease in the suspension stability of the blood cells, illustrating the physiochemical change taking place in the blood. The sedimentation test is effected more quickly than the chlorides and nonprotein nitrogen. A rapid fall in the oxygen[18] content of the venous blood occurs as a result of dehydration and slowing of the blood flow.

TREATMENT

The most important factor in the treatment of intestinal obstruction is early operation. This often is impossible when the patient is seen late with that frequent sign not of obstruction but of impending death, with vomiting, extreme dehydration, stupor, features of shock, and uremia. The presence of the toxemia demands a delay in immediate surgical intervention. Operative measures at this period would but

hasten a fatal termination. Until recently such has often been the treatment of these unfortunate patients.

It is essential first that the laboratory data be procured to determine the degree of toxemia present. The degree of intoxication will be evidenced by the degree of the variation of the blood salt, urea and carbon dioxide combining power. Such observations are an index of the profound chemical changes taking place in the tissue cells and fluids of the body. The changes in the blood can be relieved or prevented by the administration of water and sodium chloride. Distilled water, solutions of dextrose and other salts do not have such an effect. From experimental evidence Foster[19] concludes that fluids should be discontinued by mouth to prevent loss from vomiting, unless proper methods are used to prevent further chloride depletion. An adjustment of the chemical imbalance should be launched by giving intravenously, preferably, or subcutaneously, maximum quantities of 1 per cent to 3 per cent salt solution, depending on the amount of hypochloremia present, containing 10 per cent glucose. In children 10 c.c. of the solution are administered for each pound of expected weight. Reexamination of the blood chemistry should be made after a few hours. If the chemical equilibrium is not restored, the injection should be repeated until such a condition exists. In using this treatment it has been found that striking chemical improvement results, the blood urea becomes lessened, the acid base assumes normal proportions and the chloride content of the blood returns to normal.

At this time the stomach is lavaged and surgical measures are instituted. Ether is poorly withstood in acute obstruction, since it increases the shock and the amount of dehydration already present. Conversely, it has been demonstrated that spinal anesthesia has some benefit in paralytic ileus, particularly for the increased relaxation obtained, thus permitting thorough exploration with more ease and with less shock to the patient. If necessary, spinal anesthesia can be combined with a very light ethylene or nitrous oxide anesthesia to allay the mental reaction upon the patient. Following the operation glucose and sodium chloride are given in large quantities each twenty-four hours. The blood is examined daily. If the chlorides rise above normal, glucose may be given alone or the sodium chloride may be increased to 2 per cent or 3 per cent if hypochloremia persists. Gastric lavage should be carried out if retention is present.

SUMMARY

Intestinal obstruction, according to more reliable statistics, has been accompanied by a mortality rate of over 50 per cent. The chief factor in the high death rate is the delay in operative intervention with a consequent approach of toxemia. Until recently the appearance of

this toxic syndrome has heralded the advent of a fatal termination. The nature of the toxemia has, in the past, remained obscure. As the result of recent investigations it is known that characteristic blood chemistry alterations accompany this syndrome, outstanding of which are nitrogen retention, alkalosis and hypochloremia. The significance of these chemical studies is that acute upper intestinal obstruction cannot be treated logically and efficiently unless due consideration is given to chemical changes taking place in the blood.

Preoperative diagnosis has been discussed and medical treatment has been outlined which will allow the blood chemistry to reach the normal equilibrium and will permit surgical relief with some assurance of a recovery in an otherwise hopeless instance. From reports that are now creeping into the literature, it is probable that the death rate in high intestinal obstruction will be greatly lessened where proper attention is given to these preliminary and postoperative medical measures.

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ACUTE EPIDEMIC TRACHEITIS

(PRELIMINARY REPORT)

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RESPIRATORY infections, especially those of the upper tract, constitute the major portion of diseases of infancy and childhood during the winter months. With these infections there is usually an accompanying cough of varying severity. The most frequent disease of the upper respiratory tract is undoubtedly acute rhinitis which is not infrequently followed by an otitis media or postnasal infection. Perhaps the least frequent acute infection of the area above the bronchioles is tracheitis. An infection of the trachea is usually accompanied by an associated inflammation of the bronchi and bronchioles, and the condition is referred to as tracheobronchitis.[1] Practically no mention is made in pediatric literature of acute tracheitis[2] unaccompanied by bronchitis or laryngitis.

In the past few months (September, 1931, to January, 1932) I have observed 39 cases of upper respiratory infection affecting apparently the trachea alone. In fact, the frequency of it this year compared to the relative infrequency of it in the past few years, has led me to feel that we have been dealing with an epidemic form of acute tracheitis. Many southern practitioners[3] have expressed a belief that the peculiar type of severe coughs they have seen this year appear to be an acute tracheitis *per se*. It may be that the present unusual winter has been a factor in giving rise to the unusual type of cough.

The similarity of the cough of pertussis and that of acute tracheitis at times make it difficult to arrive at a diagnosis prior to the whoop observed in the former disease. In view of the valuable assistance that has been obtained in my experience from the use of white and differential blood counts and isolation of the Bordet-Gengou bacillus, those two measures were used to assist in differentiating acute tracheitis from pertussis. In a series of 78 cases of pertussis[4] in which white blood counts were employed, an average white cell count of 18,014 with a lymphocyte percentage of 62.9 per cent was found. In 12 of the 39 cases of acute tracheitis in this series, an average white cell count of 10,550 with an average lymphocyte percentage of 52.1 was noted. In the Schilling differential blood count,[5] the shift to the left percentage averaged 31.1 per cent with a shift of 25 per cent being the usual percentage in uncomplicated cases. The percentages of eosinophiles and

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monocytes were found to be 2.5 and 6.5 per cent respectively. The difference between the blood findings of acute tracheitis, and pertussis prior to the actual whoop, is that in the former the total count in uncomplicated cases shows no increase, and there is a lower lymphocyte percentage and a slighter percentage shift to the left.

In the 39 cases of this series by far the larger number of cases occurred in males, 28, or 71.7 per cent, being found in boys. The age limit varied from six months to nine years with an average age of three and eight-tenths years being noted. By far the greater number of cases were found in children over three years of age.

An unsuccessful attempt was made in 14 of the 39 cases to isolate the Bordet-Gengou bacillus which Shaw and I[6] found present in about 60 per cent of the cases of pertussis during the catarrhal and paroxysmal stages. Thus the isolation of the Bordet-Gengou bacillus gives some assistance in differentiating acute tracheitis from pertussis. No attempt has yet been made to isolate any organism other than the Bordet-Gengou bacillus.

A study of the signs and symptoms of acute tracheitis enables its differentiation from pertussis in the majority of instances. In the former the onset was found to be sudden in 76.2 per cent; hoarseness was noted in 38.4 per cent, but it was a minor symptom and was transient and of slight intensity; vomiting which occurred usually in the morning, was seen in 46.4 per cent; and in only 38.4 per cent of the cases was the onset preceded by a rhinitis. The cough in acute tracheitis is spasmodic and paroxysmal in nature like pertussis and also like that disease does not respond well to cough remedies. The cough is usually worse when lying down (especially at bedtime), arising and when the child is disturbed at night. The mucous secretion in acute tracheitis appears in from one to two days after onset of the cough; it is very tenacious in character and is coughed up only with great difficulty. As in pertussis the sticky mucus gives rise to vomiting. Occasionally the tenacious mucus gives rise to great dyspnea, and at times it is coughed up in membranelike form. The vomiting in acute tracheitis is most often noted on arising or after eating breakfast. In uncomplicated cases there is no fever or only an elevation of temperature of ½ to 1 degree. Physical examination reveals nothing of a positive nature except rattling sounds transmitted from the trachea with a few mucus râles in that area in rare instances. Careful and repeated physical examinations failed to reveal any evidence of bronchitis. Likewise there was no evidence of pharyngitis, postnasal infection or discharge, or sinus infection upon repeated physical examinations. The scant very tenacious mucus possibly accounted for failure of extension of the tracheal inflammation into the bronchioles or into the upper part of the respiratory apparatus. In the same way that acute inflamma-

tions at times seem limited to the larynx, it seems possible for infection to be limited to the trachea alone or to the trachea and possibly a portion of the larger bronchi. In fact, the moss-agate type of sputum described by Chevalier Jackson as pathognomonic of chronic tracheitis strengthens the belief that acute inflammation may be limited to the trachea.

In attempting to arrive at an early diagnosis of pertussis one is aided by history of exposure or an epidemic, of a cough progressively growing worse in spite of treatment and which is worse at night. In the early paroxysmal stage of pertussis we see suffusion of the face and vomiting following a severe paroxysm of coughing. Physical examination in pertussis like that in acute tracheitis usually gives little or no positive information. There were three cases in addition to the 39 in this series in which it was impossible to say definitely whether or not the children in question had pertussis or acute tracheitis. The blood counts resembled pertussis as did the course and response to rectal ether treatment, but there was an absence of Bordet-Gengou bacillus and of other cases of pertussis which contracted pertussis from the three in question.

From an economical viewpoint it would be unfair to consider a case of acute tracheitis as one of pertussis owing to the long period of quarantine of the latter. The period of incubation of acute tracheitis appears to be much less than that of pertussis; in all probability it varies from two to five days. Just how long this tracheal cough is infectious is not known. The duration of the cough is from three weeks to two months, usually not longer than one month.

Acute tracheitis is usually not considered an entity and is only briefly mentioned in textbooks.[2] So far as I am aware no mention has been made of an epidemic form of acute tracheitis. In only 5 of the 39 cases, or 12.8 per cent, was ordinary bronchitis noted in this series. Recurrent bronchitis was observed in 2 of the 5 cases, one of which had such severe attacks as to suggest a foreign body in the trachea or bronchi. X-ray examination failed to show evidence of any foreign body in this case.

Unfortunately response to medication was not at all favorable. The drugs used were as follows: ephedrin hydrochloride, atropin sulphate in small doses, sodium bromide or phenobarbital, syrups of ipecac or hydriodic acid, ammonium chloride, ether and olive or mineral oil by rectum,[6] and tonic doses of ultraviolet light. No attempt was made to use codeine for fear that the suppressing of a cough which had such tenacious secretion might lead to the plugging of some portion of the respiratory tree resulting in partial atelectasis. The work of Archibald and Brown[8] showed that a thin nontenacious secretion in the trachea or large bronchi is carried inward by a sudden rush of air following inspiration after a cough, but that a thick tenacious mucus as is present

in acute tracheitis is not spread by coughing. From their experimental work they feel that the former type of secretion had better be suppressed, whereas the latter type needs expectorant drugs to help expel it. Rectal ether was not effective as in pertussis[6] due probably to the failure of the ether to be secreted in the trachea and to the greater tenacity of the mucoid secretion of acute tracheitis. In the first two weeks of acute tracheitis the best, though far from desirable, results were obtained by a combination of such remedies as ephedrin hydrochloride, sodium bromide or phenobarbital, syrup of ipecac or hydriodic acid and small doses of atropin sulphate. The latter drug was used in the hope that it would possibly lessen some of the tracheal secretion. When the tracheitis cough persisted for several weeks, the ultraviolet light given two to three days apart gave the best results. When bronchitis and slight hoarseness were noted or dyspnea was present, or when cough remedies caused vomiting or failed to show any beneficial effect, inhalation of compound tincture of benzoin proved valuable. Cold air seemed to aggravate the cough in the first few weeks, whereas warm air, especially when moist, seemed to be soothing. No attempt was made to use respiratory or pertussis vaccines as it was not known with what microorganism or organisms one was dealing. X-ray greatly relieved the cough in one case and a trip to Miami, Florida, helped greatly in another.

SUMMARY

While no definite conclusion can be derived from so few cases over such a short period of time, the findings in this small series of cases makes me feel that there has been an unusual type of cough in the past few months which has the appearance of a mild epidemic and which seems to give rise to a localized inflammation of the trachea alone or that area plus the large bronchi.

In many instances diagnostic information was obtained by use of white and differential blood counts and absence of the Bordet-Gengou bacillus. The cough of acute tracheitis is spasmodic and paroxysmal in nature, is usually sudden in onset, does not become progressively worse as does pertussis and in the great majority of instances is not preceded by an acute rhinitis. Bronchitis (inflammation of the bronchioles) is not a frequent concomitant condition, and hoarseness is a symptom of transient and minor importance. The tenacity of the secretion in the trachea possibly accounts for the failure of extension to adjacent parts of the respiratory tract. Nasal or oral infections are conspicuous by their absence. The occurrence of pertussis at a previous time did not seem to have any immunizing effect in the prevention of acute tracheitis. The cough does not respond well to cough expectorants, sedatives and antispasmodic drugs. Ultraviolet light and inhalation of compound tincture of benzoin appear to give some hope of

relief from the harassing cough. Further observations and reports may lead to isolation of causative microorganism or organisms and give rise to a better plan of therapy.

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SYDENHAM'S CHOREA

The Present Status of Treatment

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THE treatment of any pathologic condition is likely to be unsatisfactory if its specific etiology is unknown. The present status of treatment in Sydenham's chorea is typical of such conditions, and the literature on this subject comprises a bizarre mass of data on almost every conceivable form of therapy. In a disease like chorea, with its innumerable variations in duration and severity, it is extremely difficult to evaluate any given method of treatment. Any form of therapy will at times appear to give striking results and lead to erroneous conclusions.

The organization of a special chorea clinic five years ago has given us the opportunity of testing different methods of treatment both as to immediate results and as to subsequent health of the children. In view of the unsettled state of opinion, we think it worth while reporting our experiences. We have not in the past, nor do we at present, hold a brief for any specific form of therapy. We have drawn our conclusions from what we consider a fair comparative test of the several types of treatment used. The patients, approximately one hundred and fifty in number, represented all grades of severity of the disease. Only one form of therapy was used in each case, and all were treated under conditions as similar as possible.

The more common forms of treatment which have been advocated in the literature may be grouped under the following heads: (1) rest, (2) drugs, (3) serums, (4) removal of foci of infection, (5) foreign proteins and vaccines, (6) dietary—general and specific, (7) prophylactic.

REST

Throughout the literature the prevailing treatment of chorea is rest. On this point there is universal agreement, and regardless of differences in additional therapy, the basic principle of all forms of treatment is complete rest, both mental and physical. We have found rest and isolation to be the most valuable of all therapeutic measures, and the earlier the disease is recognized and treatment started, the more satisfactory will be the results. At the onset even the mildest forms of the disease should be put at complete bed rest, and the child removed from

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all likely sources of emotional or mental excitation. Rarely is the parent the most suitable person to care for the patient, and often the desired mental rest can be obtained only by taking the child away from its home and placing him in a hospital or in an institution for convalescents.

The question of how long to confine a choreic child to bed is not so easily answered. In general we may say that it is wise to keep the patient in bed until all symptoms have subsided or until, after showing marked improvement, the condition has become stationary. In uncomplicated cases, this means, on an average, about six weeks. Any suspicion of cardiac involvement will, of course, prolong such a period. During this period of bed rest every effort is made to keep the child happy and contented. After the first few days he may be permitted toys and games but only those which require no fine coordination or mental exertion. We believe that few cases should be released from complete bed rest much before six weeks, even though the symptoms have abated. In this connection it is well to remember that many a child will lie quietly in bed and exhibit no choreiform symptoms until put through some mental activity. Before ordering a patient to the wheel chair, we usually ask some questions in arithmetic or geography. If the child answers these without the extremities taking on the old nervous twitching and gestures, we feel it safe to let the child up. Certain care should be exercised in this latter procedure. It is well to start with the wheel chair about one-half hour in the morning and repeat this in the afternoon. The time should gradually be extended, so that by the end of one week the patient is ready for discharge from the hospital. The ideal treatment is then the convalescent home. In some cases we have been impressed with the importance of home environment as a factor in continuing the symptoms. Particularly where the family background is the neurotic type and where the home is filled with overstimulated children, crowded together by force of poverty, we are in no hurry to return the child. In such cases where a convalescent home is beyond that patient's reach, we have seen benefit arising from a temporary sojourn with a foster parent.

DRUGS

Most observers agree that the results obtained from drugs are disappointing. There is certainly no specific, though in the past such drugs as the salicylates and arsenic have been acclaimed as such. Because of the supposed relationship between chorea and rheumatic fever the salicylates have been extremely popular. Arsenic used more or less empirically was for long the standard treatment, and from time to time good results have been reported following its use orally, intramuscularly or intravenously. Antipyrine was used extensively for many

years, especially in European clinics, but is now generally conceded to have little if any beneficial effect. Good results have been reported following injections of epinephrine in large doses and from the administration of thyroid and parathyroid extract. The use of practically every form of sedative has been advocated at one time or another. Phenyl-ethyl-hydontoin, introduced in Germany under the trade name Nirvanol, is the most recent. In most cases Nirvanol did seem to be of value in controlling excessive muscular activity. Numerous ill effects have been reported following its use, however, and though we have had no personal experience with it, we do not feel that the results obtained justify the use of a drug of such toxicity. It will suffice merely to recall the use of such drugs as magnesium sulphate subcutaneously or intraspinally; mercury; arsenic injections into the cisterna magna; electrolyzed colloidal silver intraspinally; iodine; gentian violet, methenamine, 1 per cent phenol, and sodium salicylate and arselyne intravenously; and tribromethanol anesthesia. Although claims have been made for each of these methods of treatment, none has proved its worth.

We have tried sodium salicylate and acetyl salicylic acid given in 5 to 10 gr. doses every four hours over long periods of time. When there has been a tendency toward gastric disturbances, large amounts per rectum have been used. Even in large doses, except for the fact that they may be of value as antipyretics or analgesics, their effect on the disease itself, as far as we have been able to determine, is negligible. Arsenic in the form of Fowler's solution (beginning with 2 drops three times daily and increasing to 10 to 15 drops three times a day) sodium cacodylate (1 to 3 gr. every other day) and sulpharsphenamine (10 mg. per kilogram of body weight every third or fourth day) have been tried. Other than improving the child's general nutrition, arsenic seemed to have no curative value except in possibly one case. A boy suffering from an extremely severe chorea had consistently failed to show any improvement, though at rest in the hospital ward for a period of three months. We then started him on intramuscular injections of sulpharsphenamine every third day. By the fourth injection, he showed marked improvement. He gained in weight, the violent muscular activity diminished, and his mental attitude which had been extremely pugnacious changed almost completely. Six weeks after the first injection of sulpharsphenamine he was discharged in excellent condition. Three years have elapsed. He has had one very mild recurrence and is now completely well. In the five other cases which received arsenic the course of the disease was apparently not influenced by the treatment. We were unable to obtain any striking results from adrenaline therapy or from the use of parathyroid extract. Sedatives are perhaps the most useful group of drugs. Of these luminal is fairly efficacious in, temporarily at least, controlling the excessive muscular

activity and promoting sleep. Care should be exercised in using this drug, however, as it occasionally produces a toxic reaction. Bromides are almost always of value in producing a sedative effect, and chloral has proved effective in some cases. The tolerance for these last two drugs is often markedly increased in chorea, and they frequently have to be given in large amounts before any therapeutic effect is obtained.

While occasionally some of these drugs have appeared to shorten the course of the disease, this has never been so striking as to warrant a positive statement.

SERUMS

Rosenow prepared a monovalent serum which had the property of agglutinating organisms that he had isolated from the nasopharynx and tonsils of patients suffering from chorea and reported good results from its use. This serum was tried on a series of fourteen patients. The clinical results were unsatisfactory, and the serum was not found to agglutinate specifically the organisms obtained from the tonsils and teeth of our patients.

Small used a monovalent serum prepared from a strain of streptococci isolated from cultures of the blood and pharyngeal exudate of patients with rheumatic fever and reported striking results from its use in chorea, the symptoms disappearing within a week after administration of the serum. G. Schiff tried this serum in a small series of our cases without noting any improvement in the course of the disease.

A form of therapy based on the possibility of the presence of immune substances in the blood of patients with chorea has been the intraspinal injection of autogenous serum. The intrathecal administration of horse serum has also been advocated. In none of the reports has there been what we consider clear-cut beneficial results from these treatments.

FOCI OF INFECTION

The frequent association of throat and tonsil infections with chorea have led many observers to emphasize the rôle played by focal infection in this disease. One of us (J. R. G.) studied a group of choreic children with reference to possible foci of infection, especially the teeth, sinuses and tonsils, and in one-half of the cases had the tonsils removed. While the removal of infected tonsils undoubtedly tends to improve the child's general condition, the tonsillectomy *per se* seemed to have no influence on the immediate course of the disease or on the development of recurrences. This experience is in perfect accord with that of Kaiser and others. Indeed, the immediate treatment always leaves the child worse, and any subsequent benefit is due probably more to improvement in the child's general condition than to any specific effect. It has been our impression, however, that tonsillectomy may be a factor in diminishing the severity of cardiac complications.

FOREIGN PROTEINS AND VACCINES

The use of foreign proteins in the form of whole protein intravenously, milk injections and the administration of various types of vaccines have been reported. Although good results have been claimed for each of these methods of treatment, the results in general seem to us more or less indifferent.

Recently, Sutton has reported excellent results following the febrile reaction produced by the intravenous administration of typhoid-paratyphoid vaccine. The report comes from a very conservative group, but this method of therapy has not as yet been used in a sufficiently large number of cases, under carefully controlled conditions, to justify any positive statement as to its real value. It does, however, warrant further investigation.

DIET

Dietetic treatment may be considered as part of the general treatment or as a specific form of therapy.

The general nutrition of the patient is important. The diet should consist of light, easily digested, nutritious food with a calorie value equal to that of a healthy child of the same age. Children recovering from chorea show a gain in weight. Whether this is a factor in recovery or simply a result of general systemic improvement is not certain. There is a definite indication, at any rate, that the diet should not be overlooked.

About four years ago the studies of the ketogenic diet in epilepsy suggested to us the possibility of employing some such treatment in chorea. This study was carried out in detail on fifteen children. The patients were placed on diets in which the fatty acid-glucose ratio was two to one and this ratio rapidly increased to four to one. No medication was given. Carbon dioxide content and P_H of the blood were determined at weekly intervals in addition to daily urinalyses as a means of following the degree of acidosis. All of the children showed a ketonuria by the fourth or fifth day which persisted as long as they remained on the diet. Weekly examinations of the bloods revealed no appreciable change in the P_H or any significant decrease in the carbon dioxide content. The children were on the diet for an average of forty-nine and one-half days, the shortest being twenty-six and the longest eighty-three. In only one case are we definitely able to say that we felt the diet influenced the course of the disease. This child had a severe chorea, the symptoms being present for one week before entering the hospital. He was immediately placed on the ketogenic diet, and by the end of the second week after admission all symptoms had subsided. Good results have been reported from the use of the ketogenic diet. We regret that we cannot be so optimistic as to its value.

PROPHYLACTIC TREATMENT

Is there a prophylactic treatment of chorea? If one had access to the home before the disease started, much might be accomplished. This being impossible, the next best is to supervise the children after they are discharged from the hospital. Our chorea clinic with special social service workers has been functioning for five years and enables us to do this. The children return at stated intervals, varying from every two weeks in those recently discharged to every few months in those who need less frequent examination. The children are kept at home for some weeks on semibed rest and then started on half-time schooling. During this period it is well to bear in mind that such forms of mental stimulation as competitive games, exciting stories, or motion picture "thrillers" are to be avoided. After two to four weeks of limited schooling it is reasonably safe to permit them to attend school full time.

At the end of several weeks of half-time schooling, one difficulty may arise. It is not uncommon to have the mother complain that her child is becoming more nervous again. As a rule this is due, not to a recurrence of the disease, but to the fact that the child is lonesome at home and needs something to occupy his mind. When he goes to school full time, he improves.

We have already reported that we believe such a prophylactic treatment has a definite beneficial influence upon recurrence. By keeping the children under constant supervision, it is possible to detect recurrences in their incipiency. The institution at such time of appropriate and individual treatment, rest and schooling, has spared many a child a second period of hospitalization. The results have been highly encouraging, and a more detailed report will be made at a later date.

SUMMARY AND CONCLUSIONS

What is the present status of treatment in Sydenham's chorea? Every study so far has been inconclusive. Drug therapy offers little aside from the temporary relief afforded by sedatives. The results from tonsillectomy and the treatment of other foci of infection and from the administration of serums have been disappointing. The ketogenic diet in our hands has not been of great value. Beneficial results following the use of foreign proteins or vaccines have never been satisfactorily proved. Rest and isolation still remain the most valuable therapeutic measures.

In the absence of any specific it may be well to profit by our experience in other fields of pediatrics. Prophylaxis always offers more than active treatment given tardily or blindly. Perhaps too little attention has been given to the prophylaxis of chorea, and it is possible that along such lines will be found the solution to the problem.

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BRUCELLA ABORTUS INFECTION IN INFANCY AND CHILDHOOD

Review of the Literature and the Report of a Case of a Six-and-One-Half-Year-Old Child

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THE following is the report of a case of Br. abortus infection in a six-and-one-half-year-old child, the first case observed in this clinic. The infrequency of this disease in the Northeast and particularly in New England, and the rarity of the disease in children warrant this report. A review of the literature is presented which indicates the rarity of reported cases in infancy and childhood.

There has been much confusion between Brucella abortus and Brucella melitensis. These two organisms were both discovered separately toward the end of the last century. Their similarity was not disclosed until many years later at which time they were found to be so similar that it was thought for a time that they might be identical. Recent evidence indicates that they are two closely related but distinguishable organisms.

Brucella melitensis was first isolated from a patient with Malta fever in 1887 by Bruce[1] whose name the organism bears. Its source was traced to the infected milk of goats. This disease has occurred chiefly in Mediterranean countries where goat's milk is used. From these countries several cases have been reported in children.[2, 3, 4] In the United States, infection of caprine origin has been recognized since 1903.[5] Since then it has been found not infrequently among goat-herders of the Mexican border, and sporadically in other parts of the country.

Brucella abortus was first described by Bang[6] in 1897. He recognized it as the cause of infectious abortion in cattle. That it might be pathogenic for man as well as cattle was not suggested until 1913. At this time the blood of supposedly healthy individuals was found to contain circulating antibodies for the bovine strain. Larson and Sedgwick[7] examined the sera of an unselected group of 425 children by the complement-fixation method using Br. abortus as antigen. They found that 73 (17 per cent) gave positive reactions. Further study

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of the original series of 425 by the same authors[8] revealed that those receiving milk from abortus-free herds were serologically negative while a high percentage of positive reactions was found in those fed on market milk. In one group with the same milk supply, positive reactions were found in 48 per cent. Ramsey[9] made a similar examination of 116 children and found positive complement-fixation reactions in 7. These facts presented evidence of the invasion of the human body by the bovine organism, but as yet evidence was lacking that it caused disease in man.

Evans[10] in 1918 demonstrated the close relationship between Br. abortus and Br. melitensis. These two strains were shown to be so closely related that they could be differentiated only with difficulty by the usual laboratory methods. The former was known to pass from goats to man and cause Malta fever; the latter was known to infect cattle and had been found abundantly in milk samples.

However, it was not until 1924 when Keefer[11] reported a case of Malta fever due to the abortus variety of the Br. melitensis group that the bovine strain was shown definitely to cause disease in man. Similar cases then began to appear in the literature in rapidly increasing numbers. The most recent and comprehensive survey of the disease in this country has been made by Hardy.[12] He collected 1,296 cases excluding any suspected of being caused by Br. melitensis.

REVIEW OF THE CASES OF BR. ABORTUS INFECTION IN INFANTS AND CHILDREN

Despite the rather rapidly accumulating number of cases appearing in the literature, cases of Br. abortus infection in infants and children remain comparatively rare. We have been able to find a few in the foreign medical literature in which infection of caprine origin may be definitely excluded.

In England, Williamson[13] reports a case of undulant fever in an infant twenty months old. This patient was the son of a farmer who had recently had abortion among his cattle. The infant had an undulating fever for six months with diarrhea and anorexia, cervical lymphadenopathy, palpable liver and spleen, and an initial leucopenia. Agglutination with Br. abortus was positive in a dilution of 1:250. Williamson believes this to be the first recorded case in infancy in England.

In the German literature, Fleischmann and Raddatz[14] report a ten-year-old patient with "Bang Infecktionen" in which the diagnosis was made by a positive aggultination in a dilution of 1:800. Koschate[15] in a survey of the German literature in 1930, found no recorded cases in patients younger than fourteen years, and reported a case in a twelve-year-old boy. Here the clinical picture was supported by a positive agglutination in a dilution of 1:480. Subsequently, Tobler[16] reported the case of an eight-year-old girl whose blood agglutinated Br. abortus in dilutions up to 1:2000 and gave a four-plus complement-fixation test.

Kling[17] in a series of 25 cases collected in 1928 from Sweden found the youngest to be eleven years old.

Kristensen and Holm[18] have reported 500 cases of undulant fever in Denmark. The sera of the patients in this group agglutinated Br. abortus in dilutions of

1:100 or greater. Of this series 5 were between six and nine years, and 28 between ten and fourteen years of age.

In the United States the occurrence of the disease in children was first suggested by the early work of Larson and Sedgwick[7, 8] referred to above. Despite the comparatively high morbidity which their work suggested, recorded cases in children remained few. In 1928 Kern[19] reviewed the literature and found only 36 cases in which infection by the caprine strain could be definitely excluded. Of the 36 the youngest was thirteen years old, all other cases being over fifteen. At the same time the author presented the case history of a thirteen-year-old boy with the disease. Wainwright[20] collected 51 cases of Br. abortus infection. Of these there were 5 cases during the first decade and 5 cases during the second decade. Of Hardy's total number of cases (1,296)[12] there are 4 below the age of five years, 16 from five to nine years, and 30 between ten and fourteen years, giving a total of 50 patients fourteen years or younger, or about 4 per cent of the total series. Subsequent to Hardy's report, Simpson and Frazier[21] reported 63 cases in and around Dayton, Ohio. Of this series 6 were in the first decade and 8 in the second decade. The most recent addition is the report by Anderson and Pohl[22] of 3 cases. These patients were three, six and eight years old, respectively. The agglutination tests were positive (1:320, 1:640, 1:40, respectively), and the cows supplying milk to the patients were shown to be infected.

In regard to Br. abortus infection in infants, it is often said that they are immune. Mention has already been made, above, of the British case in a twenty-month-old infant.[13] Hardy[23] refers to a patient twenty-two months old. There has recently been reported the case[24] of an infant, six and one-half months of age, who began losing weight and developed a low-grade fever with a polymorphonuclear leucocytosis. A diagnosis of undulant fever was made on the basis of a "four-plus" agglutination reaction in dilutions up to 1:640. However, there is no indication in the report as to whether this represents an infection due to Br. melitensis or Br. abortus. The youngest convincing case of Br. abortus infection which we have been able to find in the literature is that of a male infant one year old[25] who had been breast fed for the first two months of life and then put on a milk formula (Kohlbry's report). The infant had been sick with anorexia, vomiting, cough, loss of weight, increasing pallor, and an intermittent fever. On admission the temperature was 102° F. The liver and spleen were enlarged. There was secondary anemia and leucocytosis and agglutination with Br. abortus in dilutions up to 1:280.

In this connection it is interesting to note that in the series reported by Larson and Sedgwick[7] there was one infant who had been artificially fed beginning on the seventh day and whose serum showed a positive reaction with Br. abortus on the twenty-first day. It is uncertain whether this represents an active immunity established subsequent to the ingestion of infected milk or whether it might be a passive immunity conferred by a serologically positive mother. This latter has been reported in the literature.[26] Mention may also be made at this point of a case[18] in which the mother miscarried two months before term. Br. abortus was isolated from an exudate on the maternal side of the placenta. The organism could not be obtained from the fetus.

CASE REPORT

L. Q., a male child, aged six years, nine months, was brought to this clinic for the first time on September 17, 1931, with a complaint of fever for nine months. He was living with his family on a farm. Information obtained subsequently revealed that a cow purchased for the farm two years previously had had several abortions and was therefore sold. However, there followed at least three abortions in the

herd, the last about one year before this time. The boy drank the unboiled milk from this herd. The past history was negative. The present illness dated back nine weeks before admission when the child began to have fever ranging up to 104° F. with afternoon chills for about four weeks. This was accompanied by headaches, vomiting, general fatigue and occasional vague abdominal pains. A diagnosis of pyelitis was made and treatment with caprokol instituted. His condition was somewhat improved during the five weeks before admission, but the pyrexia continued, occasionally as high as 103° F.

Physical examination on admission showed a pale, well-developed and well-nourished, white, male child, not appearing very sick. There was a soft systolic basal heart murmur. The liver was felt two fingerbreadths below the costal margin, and the spleen one fingerbreadth below the costal margin. The throat was slightly inflamed. A few small lesions were present on the abdomen consistent with, but not typical of, rose spots.

Laboratory Data.—The *urine* showed no pyuria. It remained negative throughout his stay in the hospital except for the bacteriologic findings mentioned below. *Hemoglobin,* 80 per cent (Talquist); *red blood cells,* 4,450,000 per c.mm.; *white blood* cells on admission, 8,600, per c.mm., and three weeks later 8,750 per c.mm. *Differential count:* polymorphonuclear leucocytes 21 per cent, lymphocytes 61 per cent; monocytes 16 per cent, lymphoblasts 2 per cent. *Wassermann* reaction negative. *Tuberculin* test negative in a dilution of 1:1000. *Roentgenograms* of the chest showed a moderate amount of coarse flocculent peribronchial infiltration and slight thickening of the septum between the middle and upper lobes of the right lung. The day after admission, agglutination of the patient's serum with a stock culture of Br. abortus was positive up to dilutions of 1:2560. Specimens of venous blood and urine, the latter obtained by catheterization, were then cultured in dextrose hormone broth with 10 per cent of ascitic fluid. These were cultured in an atmosphere of partial carbon dioxide tension. After ten days incubation, Br. abortus organisms were found to have grown in pure culture from both the urine and the blood. On October 8, 1931, a second agglutination was performed. At this time the patient's serum agglutinated the stock culture of Br. abortus as well as the organism obtained from the blood culture in dilutions up to 1:10,000.

During the first eighteen days in the hospital the child showed a daily rise of temperature of 101° F. to 102° F., but otherwise did not seem ill. After the eighteenth day the temperature and pulse remained normal during the next nine days and he was discharged.

COMMENT

This is the only case of Br. abortus infection which has been seen up to date in this clinic. In 1913, Larson and Sedgwick[7] in a series of 425 children found 17 per cent serologically positive for Br. abortus. This high percentage of positive serologic reactions does not agree with the findings of subsequent workers. In this clinic, Guest[27] examined for Br. abortus agglutinins, serum from more than 250 children. These sera had been submitted primarily for a routine Wassermann test. Of these he found only one specimen which reacted positively through dilutions of 1:40. An agglutination of this value cannot be considered of great significance.

During the five years prior to this report (Jan. 1, 1927, to December 31, 1931) there have been 111 agglutination tests for Br. abortus

on infants and children with obscure pyrexia. Of these, the case presented here is the only one which was positive. These agglutinations do not represent routine examinations. In each instance the test was specifically requested for diagnostic purposes. This makes even more striking the low positive percentage in this clinic as contrasted with the higher percentage of Sedgwick who used sera taken for routine Wassermann tests. We realize that 111 agglutinations do not represent a sufficiently comprehensive study from which to draw accurate conclusions. However, it does suggest that, at the present time, not only is the disease rare in childhood, but also that the number of serologically positive infants and children is very small.

For comparison it is interesting to consider the percentage of positive agglutinations in several recent series which are not limited to children. In 1928 McAlpine and Mickle[28] reported that in Connecticut, 10,157 sera sent in for the Wassermann test, showed positive Br. abortus agglutinations in 63 (0.6 per cent). More recently Carpenter, Boak, and Chapman[29] found 7.3 per cent positive reactions out of 4,050 agglutination tests in New York State. McKay and McNabb[30] in Canada found of 856 sera agglutinated with Br. abortus, positive results in 17 (1.9 per cent). Welsh[31] performed agglutination tests on 2,433 sera sent from different parts of Maryland and found 133 (5.4 per cent) which agglutinated Br. abortus in dilutions of 1:25 to 1:100.

SUMMARY

1. A review of the literature of Br. abortus infection in infants and children indicates the infrequency of the disease in the younger age groups.

2. A case of Br. abortus infection in a six-year-old boy is presented in which the history and clinical course are typical, and in which the agglutination reaction, blood culture and urine culture were all positive for Br. abortus.

3. One hundred and eleven agglutinations have been done on the sera of patients where the diagnosis of Br. abortus infection was considered. Of these, the case presented here is the only positive one.

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CHILDREN'S HOSPITAL.

AN EVALUATION OF DIPHTHERIA IMMUNIZATION

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THE possibility of immunizing against diphtheria is an established fact. That such immunization can be accomplished by several different preparations and methods of administration has been amply demonstrated. However, the ideal immunizing agent has not yet been found, and considerable confusion still exists in regard to the best method of administration of both toxin-antitoxin and toxoid. An appraisal of diphtheria immunization is therefore not out of place.

Until 1923, three injections of toxin-antitoxin given in 1 c.c. doses at weekly intervals was the almost uniform routine in the United States. The work of selecting the most effective mixture of toxin and antitoxin, and the amount of underneutralization that was the safest and most efficient, as well as establishing a routine of administration, was done almost entirely by Park and Zingher and their collaborators[1-5] in and about New York. Within the last ten years numerous reports with varying results have been published. To save time and space and to make the figures easily accessible to those interested, some of these have been outlined in Tables I and II. In a previous paper attention was called to the fact that reports have shown a decreasing percentage of successful immunization by the routine of 3 injections of toxin-antitoxin at weekly intervals dropping from 90-95 per cent as reported by the Department of Health, New York City,[6] and Park, Schroder and Zingher[7] to 60 and 70 per cent in recent reports from other parts of the country, Table I.

It is well to call attention again to the possible explanation of these discrepancies. The proper underneutralization of the toxin is undoubtedly the most important factor in the immunizing efficiency of the product. The Hygienic Laboratory provides for a certain range of underneutralization. Too much free toxin is irritating to the tissues. Reactions with injections discourage its use by both doctors and lay people. The tendency of commercial houses, therefore, is to prepare the product with the maximum rather than the minimum neutralization. Furthermore the 0.1 L+ product is less stable[8, 9] than the 3 L+ or products with larger amounts of neutralized toxin, and deterioration

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TABLE I

RESULTS IN DIPHTHERIA IMMUNIZATION WITH TOXIN-ANTITOXIN

AUTHOR	YEAR	AGE GROUP	NO. OF CASES	NO. INJECTIONS T.A.T. AT WEEKLY INTERVALS	RE-SCHICK INTERVAL	PER CENT IMMUNE
Dept. Health, N. Y. C.[6]	1921	Children	201	3	3 months	95.0
Park[3]	1922	Children		3	4- 5 months	85.0 (approx.)
Blum	1922	Infants	30	3	3-15 months	100.0
Byard	1922	Infants	286	3	8 months	97.6
Park, Schroder and Zingher[7]	1923	Children	490	3-1/10 L	4 months	90.0
			304	3-1/2 L	4 months	95.0
Sears[60]	1924	Pre-school	113	3	6 months	94.0
		School children	1,594	3	6 months	62.0
Zingher[39]	1925	Children		3	12 months	82.0
Schwartz and Janney[21]						
Milwaukee schools	1925	Children	several 1,000	3		85.0
	1926	Children		3		62.0
	1927	Children		3		75.0
	1928	Children		3		69.0
Smyth and Nesbit (Gary, Ind.)		Children	1,668	3		70.0
Gute		Children	392	3		66.3
Schwartz and Janney	1930	Children	361	3		78.0
Schwartz and Janney	1930	Children	104	3 Sheep T.A.T.	6 months	71.0
Seligmann[61]	1929	Children	11,325	3 T.A.T. and T.A. floccules	6 weeks or more	71.0
Harrison[62]	1930	Children	362	3		64.0
Fuesterman[23]	1930	Children	439	3	5 years	91.5
Thelander[19]	1930	Infants	112	3	6- 8 months	75.0
Benz[22]	1931	Children and infants	1,654	3 Horse and goat T.A.T.	6 months	81.2
Volk[15]	1931	Children	336	3 T.A.T.	1 year	64.4
					2 years	85.0
Thelander	1931	Infants	77	4	6 months	92.3
Volk[15]	1931	Children	946	4 T.A.T.	1 year	74.0
					2 years	92.0
Muslow[63]	1921	Adults	31	3	3 months	75.0 (approx.)
Cooke[64]	1921	Adults	62	3	4-15 months	75.0-79.0
Hooker[65]	1924	Adults	171	3		75.0 (approx.)
Crooks[66]	1925	Adults	156	3	3- 6 months	93.6
Lintz[18]	1926	Adults	65	3		55.4
	1926	Adults		4		57.6
	1926	Adults		5		72.3
Rhoads[10]	1928	Adults	43	3	3 months to 5 years	67.2
Dick and Dick[16]	1929	Adults	100	5		82.0
Rhoads[17]	1931	Adults	87	5		64.4

TABLE II

RESULTS IN DIPHTHERIA IMMUNIZATION WITH TOXOID OR ANATOXIN

AUTHOR	YEAR	NO. OF CASES	NO. OF DOSES	AMOUNT PER DOSE IN C.C.	INTERVAL BETWEEN DOSES	RE-SCHICK INTERVAL	PER CENT IMMUNE
				CHILDREN I			
Park and Zingher[47]	1924	83	2	A and B*			75.2
		245	2	C			68.1
		401	3	A and B			94.2
		1,012	3	C			86.0
Zingher[39]	1924		2				86.4
Lereboullet†	1926	1,414	2				95.0
			3				100.0
Fitzgerald[46]	1927		2	0.5-1.0	1 month	3 months	70.0
Bloomberg, Fleming[38]	1927	208	2	0.5-0.5	1 month	4 months	68.0
		38	2	0.5-1.0	1 month	3-4 months	84.0-91.0
		32	3				94.0
Ramon[43]	1928		2			2 months	90.0-95.0
			3	0.5-1.0-1.5	3 weeks 1st 15 days 2nd	2 months	97.0-100.0
Frankl[42]	1928	163				6 months	93.8
						8 months	100.0
Kryloff (League of Nations report)[48]	1928	880	3				91.4
Silverman[50]	1929	7	2	0.5, 0.5	2 weeks	9 months	86.0
Schwartz, Janney[31]	1930	93	3	0.5, 0.5, 1.0	2 weeks		98.0
Harrison[45]	1930	426	3	0.5 each	1 week		92.0
			2	1.0 each	31 days		92.0-96.0
			2	1.0 each	42 days		99.0
Fuesterman[33]	1930	125	2	1.0 each	3 weeks	6 weeks	93.4
Benz[23]	1931	1,372	2 and 3		3 weeks	6 months	96.7
Greengard[41]	1931	100	2		1 and 3 weeks	2-6 weeks	98.0
Ray[71]	1931	333	2	1.0	10 days	3 months	98.0
		25	1			3 months	84.0
Park[49]	1932	112	3	0.5 c.c. with alum	1 week		98.2
				ADULTS II			
Maloney, Fraser[27]	1927		2	0.5, 1.0	1 month	3 months	70.0
			3				98.0
Weinfeld, Cooperstock[55]	1929	89	2	1.0	1 month	5-7 months	92.0
Dick, Dick[16]	1929		3	0.5, 1.0, 1.0	2 weeks		93.3
Cooperstock, Weinfeld[70]	1930		2	1.0	3 weeks	3 weeks	58.0
						9 weeks	79.5
						16-22 weeks	92.3
Rhoads[17]	1931	276	3 or more	Total 2.5 c.c.			81.4

*A, B, C, 3 different lots of toxoid used.
†Quoted by Greengard, note 42.

of preparations, especially when provided in larger quantities for school and board of health work, is liable to occur. Rhoads[10] demonstrated that several lots of commercial products at the time of testing were below the permissible variation granted by the Hygienic Laboratory.

Variations in the material used for immunization, however, does not explain all results; a variation in the subjects immunized must also be recognized. Zingher[3] found that the percentage of natural immunes (Schick negative without immunization), was higher in the schools where children came from congested districts than in private schools, and that it was higher in urban than in rural communities. The explanation offered for this observation was that crowding favored repeated exposure to diphtheria or to carriers, and frequent exposure even though the disease was not contracted frequently resulted in immunization. If it did not result in complete immunization, the cells were so sensitized by the contact that further stimulation, such as given by toxin-antitoxin or toxoid injections, produced a rapid response with complete immunity. Thus the immunization of the Schick positive group in congested districts was more easily accomplished than a similar group in exclusive districts. Zingher states that this variation ranged from 20 per cent to 60 or 70 per cent where the same mixture of toxin-antitoxin was used in different schools on children reacting positively to the Schick test. In some orphan institutions only 5 to 10 per cent were Schick positive with the preliminary test, the longer the children had been institutionalized irrespective of age, the higher the percentage of immunes. Of the Schick positive group fully 85 to 100 per cent of the nonimmunes became Schick negative with the first series of injections. Dudley's[11] experience in a boys' school was similar. In a group of boys entering school 32 per cent were Schick negative, of the one term boys 57 per cent, of the two term boys 60 per cent, and of the four term boys 83 per cent irrespective of age. Of the new boys who were positive and given the injections only 35 per cent were Schick negative after six weeks, while of the positive group who had lived in school for more than one year 78 per cent were negative six weeks after the immunizing injections. Hercus et al.[12] working in a remote New Zealand community where diphtheria was not endemic found that not only was the percentage of Schick positives on the original Schick testing high, but that seven months after immunization 72 per cent were still positive. He concludes that active immunization in such communities requires more time and more intensive courses of prophylaxis than areas where diphtheria has been endemic, and that statistics are of no value unless observations are made on groups with the same original herd immunity. May and Dudley[13] state that the diphtheria bacillus tends to become extinct in the immune herd. If the above hypotheses are true, then with our present control of diphtheria by isolation and immunization, the

chances of repeated contact with diphtheria are lessened, and the establishment of the necessary sensitization is less frequently produced. Consequently many of our modern urban groups of children resemble in immunity the former rural groups.

Hirszfeld[14] has not been willing to accept the environmental hypothesis as the chief factor in establishing immunity. He found a high correlation between resistance and susceptibility of parents and children and regards this association as being chiefly the result of inheritance. In support of his genetic theory, he found where the parents belong to different blood groups and react differently to the Schick test the child will show the same Schick reaction as the parent whose blood group he has inherited. It is probable that both environment and heredity play a part, and there may be still other factors concerned in the development of natural immunity. Be that as it may, allowance must be made for a changing basic immunity.

Some experiments indicate that a longer interval than a week between injections of toxin-antitoxin may give better immunizing results. Zingher[3] found in the study both of "naturally" immune children and of horses known as good antitoxin producers, that it required seven to ten days after an injection of toxin-antitoxin before a sufficient increase in antitoxin was noted in the blood to be detected by the examination of the blood serum. Those people who remain Schick positive after a first series of toxin-antitoxin injections frequently respond very quickly with antitoxin formation in the blood with one or more subsequent injections. This rapid response again probably is due to the sensitivity produced in the cells by the first series. The interval between injections, therefore, may be of greater significance in the immunity response than the number of injections. Zingher[3] reports the use of 3 injections of 1 c.c. each at two weeks' interval, but I have found no report on the re-Schicking of this group. In the series where 2 injections of 1.5 c.c. each were given at one week's interval the percentage of immunes was lower than in those receiving 3 injections at weekly intervals. Very few reports on variations in either dosage or interval with toxin-antitoxin have appeared in the literature. The reason for this is that the original reports with 3 injections were so satisfactory that there seemed little need for variation, and furthermore when less favorable reports began to appear, the French, 1924, were reporting excellent results with anatoxin, consequently instead of varying the old routine, anatoxin or toxoid has been substituted. Volk[15] gave 4 injections to 946 children—after one year 74 per cent were immune, after two years 92 per cent, as compared with 64 and 85 per cent with 3 injections. A number of investigators (personal communication) who have given 4 doses to children instead of the routine three, have had good results, but unfortunately these have not been published. In adults, as seen in Table I several authors have reported a high percentage of immunes with four

and five injections as well as with repeated courses to persistently Schick positive adults. Dick and Dick[16] report 24 per cent immunity in nurses who had received the routine immunization of three injections in other institutions. With five injections they increased this percentage to 82 per cent. Rhoads[17] reports the immunization of 64.4 per cent of 107 Schick positive nurses (in the Cook County Hospital) by five doses of toxin-antitoxin at weekly intervals totaling 4.5 c.c. of the mixture. The experience of other authors has been similar (Table I), though it has been uniformally observed that some individuals are very refractory to immunization. Lintz[18] reports a nurse, Schick positive after six courses consisting of three injections each of toxin-antitoxin.

At the Health Center conducted by the American Association of University Women since our report[19] of last year until January 1, 1931, four injections of toxin-antitoxin 1 c.c. each at weekly intervals have been given. All of this group received their injections under eighteen months of age. To date 77 children have been re-Schicked after intervals of from five to six months. Of these 77, six were positive. In other words 92.3 per cent were immunized with 4 injections of toxin-antitoxin 1 c.c. each, at weekly intervals, as compared with 70.1 per cent in 115 cases reported last year with three injections of the same material. Furthermore, the six positives were graded as one- and two-plus only, there being no strongly positive Schick reactors. Whether better results with 4 injections are due to the interval between the first and fourth dose, or to the number of injections is an interesting speculation. If toxin-antitoxin is to be used as an immunizing agent, it will be a worth-while investigation. Indications are that toxin-antitoxin will be replaced by toxoid. The general opinion is that even if given the same dosage at the same interval toxoid will prove more efficient for immunization than toxin-antitoxin.

The French did not adopt toxin-antitoxin to any extent, and among the reasons given was the danger of severe reactions. In view of the extensive use of this material in the United States and other countries with practically no reports of severe reactions, this does not seem a just objection. Park and Schroder[20] investigated this subject and found only three accidents. First, in Dallas, Texas, a mistake was made and injections were given with toxin-antitoxin having an excess of free toxin. Second, in Vienna, the toxin was put up similarly to the toxin-antitoxin mixture resulting in the use of the wrong material. Third, in Australia, it was put up without antiseptic and resulted in the use of contaminated material. In Boston minor reactions occurred caused by the use of frozen material, freezing causing dissociation. The reactions reported[19] in our group last year were also comparatively mild, and investigation disclosed that partial dissociation had occurred. This may have been due to storage in the modern electric refrigerator. Studies of the effect

of low temperature on dissociation of toxin-antitoxin mixtures have been made by White and Robinson[21, 22] and Anderson and Leonard.[23] Other factors involved in dissociation have been reviewed by Schmidt.[24]

Of greater significance than reactions to the injections, is the possibility of sensitization to horse serum by the immunization procedure. This question has produced considerable controversy. That sensitization does occur in spite of reports to the contrary can hardly be doubted with the evidence on hand. Hooker[25] reported 27 per cent increase in sensitivity after toxin-antitoxin administration as determined by skin tests. Park[26] concluded that sensitivity was slightly increased but not enough to be significant. Bauer and Wilmer[27] and Spicer[28] noted no increase. Stewart[29] reported seven cases of severe serum disease following the injection of horse serum in previously immunized patients. The most detailed report is that of Gordon and Creswell.[30] They give the following figures for patients treated with diphtheria antitoxin:

	CASES	PERCENTAGE OF SERUM REACTIONS
Total	1,556	30.2
Previous toxin-antitoxin	283	75.5
Previous serum	90	41.1
No previous injections	1,183	19.1

Seventy-three and five-tenths per cent having received toxin-antitoxin reacted with serum disease, while only 19.1 per cent having no previous toxin-antitoxin reacted thus, and 41.1 per cent having had previous serum injections with subsequent injections had serum disease. These figures indicate that repeated injections increases the susceptibility to serum disease.

The series at Children's Hospital brought up to date is as follows for patients receiving diphtheria antitoxin:

	NO. OF CASES	PERCENTAGE OF SERUM REACTIONS
Total	200	15.0
Previous toxin-antitoxin	15	100.0
No previous toxin-antitoxin	185	8.1

That these varying results are due to different interpretations by the investigators is possible. It is more probable that the toxin-antitoxin mixture used in the preliminary immunization affects results. Anderson and Leonard[23] observed that variations in toxicity on freezing was influenced by the type of antitoxin used in the preparation of toxin-antitoxin mixture, the less refined substance giving a greater dissociation. If horse serum of low antitoxic concentration were used, more horse serum would be introduced, and this might vary the type or degree of sensitivity produced. The Cutter Laboratory, which has furnished us with most of our material, uses a highly antitoxic serum for the toxin-

antitoxin preparation in order to reduce to a minimum the amount of horse serum. Our series of serum disease does not indicate that small amounts lessen the tendency to sensitize.

To avoid the danger of sensitization to horse serum, most commercial houses are preparing toxin-antitoxin mixture with sheep or goat serum. The immunizing power of this mixture is apparently the same as that of the old preparation. Schwartz and Janney[31] report 71 per cent immune, Benz[32] 81.2 per cent, and Fuesterman[33] 85.6 per cent. Although few untoward results have followed horse serum sensitization by previous toxin-antitoxin administration, the fact that sensitization may occur constitutes a legitimate reason for seeking some other immunizing substance.

Sensitization to horse serum by the injections does not endanger the person to further injections of toxin-antitoxin mixture. This has been amply shown by the work of Zingher[3] and the frequent reports of repeated series of injections by the various authors herein quoted. The amount of serum in the mixture is not sufficient to constitute a shock dose, even though the patient is sensitive to horse serum.

In 1922 to 1924, Ramon[34] introduced toxin modified with formalin under the name of anatoxin. Since then numerous investigators in France, Canada, and the United States have carried on experimentation with it, or with a slightly modified form. It is known either as anatoxin or toxoid. The French workers[35] found that an interval of from two to three weeks between the first and second injections allowing for the establishment of cell sensitivity produced better immunizing results than a shorter interval. The established routine in France is now a preliminary dose of 0.5 c.c., followed after an interval of three weeks by 1 c.c. and fifteen days later either 1 or 1.5 c.c. Over a million have been immunized by this procedure and practically all reports give from 90 to 100 per cent immunity. In Canada, Defries,[36] Moloney and Fraser,[37] and Bloomberg and Fleming[38] early used two doses of 0.5 each at intervals of three weeks to one month and report 70 to 80 per cent immune. Canada since then has adopted quite generally three doses of 0.5, 1.0 and 1.0 c.c. at intervals of three weeks between the first two injections and two weeks between the second and third. In this country Zingher[39] reported 86.4 per cent immune with 2 doses and 98 per cent with 3 doses of 0.5, 0.5 and 1 c.c., at two weeks' interval. A great many workers in the United States are using 2 doses of 1 c.c. each at three or four weeks' interval. In fact there seems to be no tendency toward any uniformity of administration, and one method apparently is not much superior to another, since almost all report 90 per cent or more immune. Various commercial houses have recommended doses as follows:

2 doses, 1 c.c. each, one month's interval.
2 doses, 1 c.c. each, three weeks' interval.
3 doses, 1 c.c. each, one week's interval.
2 doses, 1 c.c. each, six weeks' interval.

In San Francisco the following methods have been used by various pediatricians, and in various clinics:

1 c.c., 3 doses, three weeks' interval.
0.5, 0.5, and 1 c.c. at two weeks' interval.
0.5, 1 and 1 c.c. at two weeks' interval.
0.5 and 1 c.c., 2 doses, three weeks' interval.
1 c.c., 3 doses, one week's interval.

Park and Schroder[40] have reported on a series of 112 cases treated with toxoid to which alum has been added, with 3 doses of 0.5 c.c. each they obtained 98.2 per cent immunity.

The chief objection to toxoid is the reaction produced in older children and adults. Attempts have been made to minimize this. It has been found that those reacting with a marked pseudoreaction to the Schick test are liable to react to toxoid, but this method does not determine all reactors. Burke[41] in an extensive immunization campaign in Canada eliminated practically all bad reactions by testing with diluted toxoid before administering the full dosage. If the reaction with the test was greater than 10 mm. in diameter, doses of 0.1, 0.2 and 0.5 c.c. were given instead of the usual 1 c.c. dose. Diluted toxoid for the test is supplied with the package of toxoid by many commercial houses. Burke[41] also noted that reactors ran in families and advised testing younger children in families where older members reacted badly to toxoid. It is quite generally accepted that under six years reactors are few. In adults some very severe reactions have been noted. One nurse at Children's Hospital after 1 c.c. of toxoid reacted with a temperature up to 104° F., and a very painful swollen arm which incapacitated her for several days. It is interesting to note that following this injection she was Schick negative for a year, but since then has reverted to a slight positive again. At present nurses are given small doses of 0.1, 0.2, 0.5, 0.5, 0.5 c.c., 5 doses at two weeks' interval, trying to eliminate or minimize reactions. To date 68 nurses have been given such a series. Of this group 18, or 26.5 per cent, had some reactions. Two of these were severe, characterized by local swelling and edema and generalized ill feeling with fever. The others were less severe but sufficient to make the arm painful. Those reactions often occurred even with 0.1 c.c., and did not appreciably lessen with subsequent doses. Of these nurses 10 have been re-Schicked at three and six months' intervals. Three who were negative at three months had reverted to a one-plus positive at six months. The other 7 were Schick negative on both tests. Rhoads[17] gave nurses a total of 2.5 c.c. toxoid divided among three or more doses depending on reactions. Of the 276 retested 81.4 per cent were immune.

The duration of immunity has been tested in several instances. Time has not elapsed to permit very positive statements in regard to immunity produced by toxoid immunization. Ramon and Helie,[42] however, quote re-Schicks by Roubinovitch at intervals of eight, twelve, nineteen, twenty-three, twenty-four months and four years with a persistence of the Schick negative state. Frank et al.[43] retested 57 of 163 children immunized by toxoid and found in 14 per cent the immunity did not last more than six months. Greengard[44] retested 13 at intervals of three to ten months; of this number one child who was inoculated at two weeks and was Schick negative four weeks later was strongly positive again at six months. Park[2] states that in at least 90 per cent of children immunized by toxin-antitoxin, immunity lasts for six years and that there apparently is no difference between the naturally and the artificially immunized. Later Park and Schroder[20] retested 18 infants after seven years and found them still immune. In adults, naturally immunized, 93.4 per cent retained immunity after eight years. In the artificially immunized adults there was a slightly greater tendency to revert to the Schick positive state, 75 per cent retaining their immunity after eight years. Parish and Okell[45] found that of 533 children who were Schick negative without immunization 1.1 per cent were positive on re-Schicking at intervals of one to seven years. In 490 children where the Schick positive was changed to the Schick negative state by immunization, 5 per cent reverted to the positive state. The percentage of error is not mentioned in these figures. There is some evidence that those refractory to immunization, that is requiring 2 or more courses of toxin-antitoxin to produce a negative Schick also revert to the Schick positive state more readily than either those developing a natural immunity, or those responding to the first course of injections. With subsequent injections, however, they respond very quickly with antitoxin formation.

Lowenstein's immunizing ointment has not been tried extensively in this country. A cursory survey would indicate that about 70 per cent of the children so treated become immune. These figures, being early reports, may be too optimistic. Kegel and Gasul[46] report 55.3 per cent immune six weeks after the third inunction, and state that Baar and Louey obtained about 70 per cent immunity. Abt and Feingold[47] report 70.9 per cent thus immunized. European workers[48] have also attempted immunization by mouth and nose with varying results.

A review of diphtheria immunization is not complete without at least some reference to the reliability of the Schick test and the necessity of making a negative Schick reaction in every treated case the completion of the immunization procedure. Park[2] makes the statement: "The evidence that antitoxin is the only substance in the body that can prevent the toxin from acting is so conclusive that we can assume that a nega-

tive Schick test means the presence of sufficient antitoxin to neutralize it. The debatable points are: Is the minimum amount of antitoxin which is required to give a negative Schick reaction sufficient to prevent absolutely the development of diphtheria? If not, is it sufficient to prevent a toxemia?'' From the extensive experiments in New York by Park and Zingher the above questions, after allowing for a small percentage of error which is present in all experimental procedures, can be answered in the affirmative. Moloney and Fraser[37] found that Schick tests and blood titrations compared favorably. Rhoads[17] found that of 165 nurses with negative original Schick tests, two had moderately severe attacks of diphtheria while on duty in the contagious disease hospital. Inasmuch as there was little chance of error in reading the Schick, he concluded that the material was not potent. He therefore tested Schick material from seven commercial laboratories, one from a state board of health and one made in the hospital laboratory. The preparations were diluted as directed and tested on the same persons within twenty-four hours after dilution. Two of the commercial products gave negative reactions on all persons tested, while the other seven gave positive results with varying degrees of size and brightness. Since then he has been using Schick material prepared in his own laboratories. Kellogg and Stevens[49] found that in natural immunes the Schick test and blood titrations (Kellogg test) were confirmatory, but in the actively immunized 20 to 25 per cent of the Schick negatives showed a lack of antitoxin by the Kellogg test. They concluded that these people were latently immune, inasmuch as an injection of toxin-antitoxin would quickly raise the antitoxin content of the blood. It is essential that the sources of error in Schick testing be understood and avoided. (1) The toxin must be of the proper strength; (2) it must be freshly prepared as it deteriorates rapidly; (3) the test must be done intracutaneously, care being taken not to inject it subcutaneously. In very young children with a thin skin it becomes an art to get the material between the layers of the skin instead of underneath. (4) A control test should always be run; (5) testing should always be done on a group of children, or a known positive used as a control to check the potency of the toxin. This will avoid errors such as noted by Rhoads.[17] New diluents for Schick toxin are being studied and tested.[50-53] Some of these are on the market. At the Children's Hospital the past year, the old material and the new have been compared by applying both tests to 29 children and 30 adults. Generally the new material gives a reaction that is less discreet, slightly paler, fades more rapidly and does not leave a stain. In the 29 children there was a real discrepancy in only one instance, the old material giving a positive Schick and the new diluent a negative. In one case the new diluent had a marked pseudoreaction when the old did not. In 3 eczematous patients and 1 child who had had antitoxin there were no confusing

reactions. Seven children had had previous T.A.T., in one of these a marked pseudoreaction occurred with the new diluent and its control. In the 30 adults, there were 2 discrepancies, in both cases the new diluent gave negative Schick reactions when the old gave positive. Two cases also gave marked pseudo- and control-reactions with the new diluent. Moloney and Taylor[54] report on a modification of the diluent, by reducing greatly the peptone. The results warrant the opinion that the preparation of a stabilized Schick toxin equally as good as the old material is not far distant.

It is quite uniformly agreed and practiced to immunize children under six years of age without a preliminary Schick test. The number of positives in the younger age groups justifies it, and it simplifies the procedure of immunization.

A persistence of a positive Schick after 3 injections means lack of immunity to diphtheria. Reports in the literature furnish ample evidence of this. Schwartz and Janney[31] report nine out of 29 cases of diphtheria having had previous toxin-antitoxin injections. Rhoads[10] reports 29 cases with previous toxin-antitoxin, Weinfeld and Cooperstock[55] 10 cases in nurses in twenty-six months, all having had the injections one to three years previous. Gordon and Creswell's[30] series include diphtheria in previously immunized cases, and the above-mentioned series of serum disease includes 15 cases with diphtheria following toxin-antitoxin injections, one of which proved fatal. It is significant that in none of these had a Schick test been made. Grierson[56] of Edinburgh reports on 98 children who had been notified as suffering from diphtheria after having been Schick tested or given toxin-antitoxin or both, and of these 98, 58 were diagnosed as clinical diphtheria and 40 as bacteriologic diphtheria. Of the 58 clinical cases, 24 had been Schick tested only, of these 18 were positive, 2 doubtful, and 4 negative. Another 22 cases occurred in children Schick positive, and given 3 injections of toxin-antitoxin, but not re-Schicked. Two mild cases of diphtheria occurred in children Schick positive, given toxin-antitoxin and on re-Schicking found negative. Park, Schroder and Zingher[7] in 180,000 children in New York found four times as many cases of diphtheria in the Schick positive as in the Schick negative groups, and diphtheria was less severe and often questionable in the Schick negative group. Inasmuch as 5 to 25 per cent revert to the Schick positive state after being Schick negative, it is not surprising that cases of diphtheria should occur in this group. Those reported have been mild. It is probable that diphtheria infection in these cases stimulates the production of antitoxin just as subsequent injections of toxin-antitoxin or toxoid do in similar cases. On the other hand, those who remain Schick positive after the routine immunization procedure if contracting diphtheria will not necessarily have a mild case. They may be particularly poor anti-

toxin producers and therefore have a severe infection. Fitzgerald and his coworkers[57] report on the incidence of diphtheria in 16,829 Toronto school children who had received 3 doses, and 8,994 having received 2 doses of toxoid. In those receiving 3 injections, 3 cases of diphtheria occurred instead of an expected incidence of 222 or 90 per cent reduction in incidence. In those receiving 2 doses there was a reduction of 74 per cent.

COMMENTS

It is evident from the above survey that due to the number of variables introduced by each investigator it is impossible to make any valuable comparative studies from the reports in the literature. It would seem possible that methods could be standardized. It would facilitate investigation if a few of the following rules be adhered to:

(1) Uniformity of preparation of material by the commercial houses.
(2) A uniform interval for re-Schicking.
(3) A uniform interval in reading the Schick.
(4) A record of the type of Schick material used.
(5) A record of the ages of those immunized.
(6) A record of whether or not a preliminary Schick test had been made.

This would leave as variables the dosage and interval between the injections. If a number of series were run in this way, keeping either interval or dosage constant, the simplest and most efficient method could undoubtedly be agreed upon. The problem of reactions in older children and adults preferably should be solved by the preparation of a mixture that does not produce reactions—this may not be possible. The subject is receiving attention. The work of Glenny[58] and Swyer[59] on toxoid-antitoxin floccules is at least encouraging.

At present it would seem logical to follow the method adopted by the French after their large experience, or a slight modification of their method. If to children under six years, 3 doses be given at either two or three weeks' interval, the first consisting of 0.5 c.c. and the remaining two of 1 c.c. each, probably a maximum immunity would be attained. The preliminary dose of 0.5 c.c. would pick reactors, but in most instances avoid annoying reactions. If, on the other hand, reactions occurred, the second and third dose could be modified to meet the need. Americans are probably too much creatures of routine to adopt the three weeks' and fifteen days' intervals of the French. In older children and adults, the preliminary testing probably to date is the best method of picking reactors.

SUMMARY

1. Three injections of toxin-antitoxin mixture given in 1 c.c. doses at weekly intervals renders immune at the end of six months about 70 per cent of children and less than this of adults. Four doses of 1 c.c.

each given at weekly intervals probably immunizes about 80 to 90 per cent of children. Five doses of 1 c.c. each at weekly intervals immunizes about 85 per cent of adults.

2. Toxin-antitoxin properly underneutralized and stored gives practically no local or general reactions.

3. Toxin-antitoxin mixture is capable of sensitizing individuals to horse serum.

4. The substitution of sheep or goat serum for horse serum in the preparation of toxin-antitoxin mixture has been done. The percentage immunization with this is about the same as with the horse serum toxin-antitoxin.

5. One series of toxin-antitoxin injections does not endanger the individual to later injections of toxin-antitoxin inasmuch as the total amount of horse serum in 1 c.c. of mixture is not sufficient to produce shock even if hypersensitive to horse serum.

6. Immunization with toxoid by any method of 2 or more injections of 1 c.c. each produces 90 to 100 per cent immunity in three to six months.

7. Reactions in older children and adults are the chief objections to the use of toxoid as an immunizing agent.

8. Duration of immunity whether naturally or artificially produced is good. It is apparently more lasting if immunization is done in childhood than in adult life. There is some evidence that the individuals more resistant to immunization revert to the Schick positive state more readily.

9. The Schick test, properly carried out, is a reliable indication of immunity to diphtheria and should be considered part of the immunization procedure.

10. Individuals Schick positive in spite of the routine immunization procedure can and have contracted diphtheria.

11. A standardization of methods of immunization is desirable.

12. The ideals still to be reached are (a) an agent that gives no reactions in any age group; (b) an agent that immunizes close to 100 per cent; (c) a simpler method less subject to error for testing for immunity.

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ANEMIA OF THE NEWBORN IN THREE SUCCESSIVE SIBLINGS

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THE occurrence of a severe anemia without discoverable cause, developing at birth or within the first two weeks of life, has been reported with increasing frequency in recent years. Summaries of these reported cases and additional contributions have been made during the past year by Abt,[1] Pasachoff and Wilson,[2] and in the German literature by Stransky.[3] The latter, in his summary, includes one case not found in the American literature, which presented numerous erythroblasts and myeloblasts in the blood picture and ended fatally.[4] There is also the brief report of a case by M'Neil[5] who used liver extract intramuscularly, apparently with good results.

In the twenty reported cases there is no history of this type of anemia having occurred in more than one sibling, and its development in three consecutive babies of the same parents is of unusual interest.

REPORT OF CASES

Both parents are young adults whose general health has been excellent. During pregnancies the mother's health has been satisfactory, and she has done the greater part of the housework until late in the gestation period. Her diet was well balanced except for a limited amount of milk due to a distaste for this food. She has always eaten freely of vegetables, however, particularly those which are high in mineral content. She disclaims any excessive bleeding during the menstrual periods, and except for postpartum hemorrhages as subsequently noted no history of hemorrhagic diathesis was elicited. The familial occurrence of any disease was denied.

In 1926 a stillbirth occurred. The baby weighed 9.5 pounds and was thought to have been a month overdue. Dr. R. J. Anderson, the attending physician at this and subsequent deliveries, remarked at the extreme pallor of the baby at the time of delivery.

Blood Wassermann and Kahn tests of both parents were negative.

First Baby.—A female infant; full term; normal delivery. Birth weight was 8 pounds, 9 ounces. Moderate jaundice was noted on the first day and gradually disappeared during the first week. On the fourth day the baby was noted to be "anemic looking" and although no apparent bleeding had occurred and examination of the stools for occult blood was negative, 30 c.c. of whole blood from the mother were given intramuscularly. The following day the pallor was more severe, and 75 c.c. of the mother's whole blood were given intravenously. A second transfusion of 70 c.c. of the mother's blood was given again the next (sixth) day. The bleeding and coagulation times were prolonged to 7 and 11 minutes, respectively,

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on the day the anemia was first noted. After the second transfusion, at which time the jaundice had disappeared, the bleeding time was 3 minutes and the coagulation time 7 minutes.

The baby had lost 12 ounces in weight during the first four days but after the sixth day gained steadily and had regained her birth weight on the fourteenth day. The skin and mucous membranes continued to be very pale, and a bedside note at this time compared the color of the skin with that of old ivory. No vomiting occurred and the temperature was never over 99° rectally. Spleen and liver were not palpable. The baby was taken home on the fourteenth day. Unfortunately, no blood examination was recorded during the early newborn period.

The baby gained satisfactorily at home from the second until the fourth weeks, then began to vomit and refuse food, had difficult respiration, and lost weight rapidly. She was seen by us when six weeks old and in addition to the extreme pallor (R.B.C. 830,000), was found to have a huge spleen which extended over to the midline and down to the crest of the ilium. Her respiration was grunting in character and somewhat increased. Examination of chest was negative. Temperature was 99° F. rectally. During the sixth week she received two more transfusions of 120 c.c. and 90 c.c. of the mother's whole blood. For two weeks following these transfusions no increase in the anemia occurred and the spleen became definitely smaller. Vomiting stopped and she was given vegetable water, beef juice, raw egg yolk and orange juice, in small amounts, in addition to the formula of standard lactic acid milk. She gained weight for several weeks. During the ninth and tenth weeks pallor again became more severe and vomiting recurred, so that another transfusion of 100 c.c. of the mother's whole blood was given. From this time on she made satisfactory progress and when four and one-half months old she had an essentially normal blood picture, and the spleen was no longer palpable. At the age of four years she appears to be perfectly normal.

Second Baby.—Almost fifteen months elapsed between the births of the first and second baby. Three months prior to the delivery of the second baby the mother was taken to the hospital because of labor pains which subsided a few hours after admission. Induction of labor was attempted at this time as the baby was thought to be full term, but the procedure was unsuccessful, and the mother did not deliver until three months later.

The baby was a male infant, weighing 7 pounds, 12 ounces. The mother had a profuse hemorrhage after being returned to bed from the delivery room, and another hemorrhage three nights later. In spite of the resultant weakness and anemia she had recovered sufficiently by the tenth day postpartum to be allowed up and was discharged from the hospital on the twelfth day. Three weeks postpartum she was still quite pale and a blood examination revealed a hemoglobin of 43 per cent (Dare), R.B.C. 2,790,000, W.B.C. 5,000, with a normal differential count. She recovered fully during the following two months.

The infant lost 13 ounces within the first forty-eight hours. Neither jaundice nor fever occurred. On the fifth day an edema of the scrotum was noted, and occasional projectile vomiting occurred during the second week. Toward the end of the first week a moderate degree of pallor had developed and by the tenth day this had become quite marked (R.B.C. 1,152,000, Hg. 45 per cent), and the baby was inactive and cried very little. He nursed well, however, and took two ounces of a supplementary feeding of standard lactic acid milk to which egg yolk was added on the eleventh day. After the fifth day he gained steadily and when discharged on the thirteenth day weighed 7 pounds, 14 ounces, or two ounces more than his birth weight.

Because of the increasing pallor (Hg. 22 per cent) and projectile vomiting the baby was returned to the hospital when three weeks old, and 175 c.c. of the father's

citrated blood were given intravenously. Following the transfusion the projectile vomiting stopped and he again started to gain weight. The pallor recurred to a considerable degree during the fourth week, and the spleen, which had not been palpable became moderately enlarged and could be palpated for several weeks. From the fourth week on recovery was uninterrupted. When thirty-two months old he appeared to be perfectly normal except for a convergent squint.

Third Baby.—There was an interval of thirty-one months between the birth of the second and third baby. During the third pregnancy the mother's health was good, and, although her appearance did not suggest an anemia, a preparation containing liver extract and beef hemoglobin was prescribed and the necessity of a well-balanced diet emphasized by the attending physician.

She entered the hospital in active labor and following a normal delivery again had an unusual amount of bleeding which was controlled by massage of the uterus and the use of pituitrin. Four days postpartum a blood examination showed a hemoglobin content of 45 per cent, R.B.C. 3,340,000, W.B.C. 12,050, of which 63 per cent were polymorphonuclear cells, 23 per cent lymphocytes, 1 per cent large mononuclears, 1 per cent metamyelocytes and 12 per cent band (Staff) cells. On the twelfth day she was sufficiently recovered to leave the hospital.

The baby, a girl, weighed 8 pounds, 11 ounces at birth and lost 12 ounces in weight during the first four days. She then began to gain steadily and her weight reached nine pounds on the twentieth day. There was no apparent anemia during the first few days of life, and blood examination on the fourth and seventh days showed a fall in the red blood cell count and hemoglobin which was scarcely appreciable. Between the seventh and tenth days, however, an anemia became very apparent and the hemoglobin fell from 78 per cent to 47 per cent with a reduction of the R. B. C. from 3,7000,000 to 2,200,000. There was no discoverable loss of blood during this time, no fever, the spleen could not be palpated and the liver was not enlarged. A transfusion of 150 c.c. of citrated blood was given on the eleventh day. Jaundice and slight fever developed on the following day, and the beneficial effect of the blood was not long sustained so that a second transfusion was done six days later. The spleen had become palpable the day following the first transfusion and thereafter gradually enlarged and within a week extended almost to the crest of the ilium. Routine examination of the urine on several occasions was negative and urobilinogen was not increased.

Occasional projectile vomiting occurred during the second week. This subsided after the second transfusion, and the baby made good progress for a time. During the fourth week the anemia and vomiting recurred and she was again transfused with 120 c.c. of citrated blood from a paternal uncle. A severe reaction occurred with jaundice, diarrhea and a temperature of 103.6° rectally. A fourth transfusion of 125 c.c. of citrated blood was necessary four days later and no reaction occurred. During the fifth week cod liver oil and orange juice were added to her diet of whole lactic acid milk, and she was also given syrup of ferrous iodide, 30 drops daily. From the fifth to the ninth week progress was very satisfactory, and the spleen diminished in size as the infant's general condition improved. When four months old her father reported her to be in excellent health.

SUMMARY OF CASES

The cases reported fall into the class of severe anemia of the newborn in which there is no evidence of unusual blood loss and no signs of infection. The outstanding clinical signs in these cases were marked pallor which developed between the fourth and tenth days after birth,

a tendency to vomit, enlargement of the spleen which occurred only after the anemia was well developed, and the absence of fever. The infants took their feedings and gained surprisingly well until the pallor reached a certain degree of severity when lassitude and vomiting, the latter often projectile in character, made their appearance. These latter symptoms were regarded as indications for blood transfusion. We feel that transfusions in our cases were the means of sustaining life until the hematopoietic processes began to function normally. This apparently spontaneous improvement began during the eleventh week in the first baby, during the fourth week in the second, and during the seventh week in the third baby.

TABLE I

BLOOD EXAMINATIONS IN CASES REPORTED

	R. B. C.	HG.	W. B. C.	NORMOBLASTS	RETICULOCYTES
First Baby					
Sixth week	830,000	Less than 20%	29,000	Many	
Two transfusions of 120 c.c. and 90 c.c. of mother's whole blood.					
Seventh week	3,020,000	45—Dare			
Eighth week	3,044,000	45—Dare			
Ninth week	2,170,000	38—Dare 36—Newc.	7,200	Occasional	7.1%
Tenth week. Marked increase in pallor and vomiting, 100 c.c. blood given.					
Eleventh week	3,070,000	52—Dare	8,400		
Twelfth week	3,080,000	39—Dare 37—Newc.	8,500	Occasional	
Fourteenth week	2,970,000	54—Dare 47—Newc.	5,300	Occasional	10.3%
4½ months	4,530,000	76—Dare	10,200		
20 months	4,310,000	79—Dare	5,100		
Anisocytosis and poikilocytosis very marked in first examination. Fairly marked until four and one-half months.					
Second Baby					
Second week	1,520,000	45—Dare	20,000		3.0%
Third week	1,150,000	22—Dare	15,400	Occasional	17.0%
Transfusion of 175 c.c. of father's blood.					
Fourth week	1,740,000	40—Dare	9,200		16.0%
Sixth week	2,720,000	45—Dare	8,000	Occasional	
Eighth week	3,300,000	49—Dare	7,800		
Anisocytosis and poikilocytosis fairly marked until sixth week.					
Third Baby					
Fourth day	4,080,000	83			
Seventh day	3,730,000	78	7,900	Occasional	
Tenth day	2,220,000	47			
Transfusion of 150 c.c. citrated blood.					
Fourteenth day	2,700,000	59			
Sixteenth day	1,760,000	42	21,700	Occasional	5.8%
Transfusion of 120 c.c. of citrated blood.					
Eighteenth day	3,080,000	65	10,200		4.5%
Twenty-eighth day	1,610,000	40	6,400		
Transfusion of 120 c.c. blood, reaction with fever, diarrhea, jaundice.					
Thirty-second day	2,160,000	35	14,000	Occasional	8.8%
Transfusion of 125 c.c. blood. No reaction.					
Thirty-fourth day	2,400,000	46	15,700	Occasional	
Sixth week	2,040,000	38	18,000		
Ninth week	2,980,000	50	7,000		
Anisocytosis, poikilocytosis marked until after sixth week.					

The abnormalities of the blood of the infants were confined to the erythrocytopenia, low hemoglobin, marked anisocytosis and poikilocytosis, polychromasia and an inconstant leucocytosis, with an essentially normal differential count except for an occasional myelocyte and myeloblast. Immature blood cells were not present in any considerable number except at one examination when the red blood cell count fell below one million. The increase in reticulocytes during the third and fourth weeks in the second baby was coincident with the clinical improvement which became apparent during the fourth week. In the other two babies the lower reticulocyte counts might be considered consistent with the more prolonged state of anemia that was observed clinically.

DISCUSSION

It is interesting to note in connection with the absence of any considerable number of immature blood cells in our cases that Stransky[3] has divided this type of anemia into two groups, basing his distinction upon the presence or absence of immature blood cells in the circulating blood. He observed that the greatest number of immature cells occurred in the fatal cases, while those which recovered showed comparatively few embryonal forms. In two of the three fatal cases reviewed by him there was marked hematopoietic activity in the liver, spleen, lymph glands and bone marrow, unusual activity in the third case being limited to the bone marrow. He concludes that the severity of the disease is dependent upon the degree of involvement of the hematopoietic system, that in the fatal cases there is probably a faulty or incomplete development of the organs of hematopoiesis, while in the cases of lesser severity there is a temporarily insufficient hematopoiesis. Schleussing[6] previously had made a somewhat similar observation in discussing his fatal case and expressed the belief that "the blood forming apparatus during its development was overtaken by birth." He was of the opinion that Susstrunk's fatal case was similar to his own in this respect in spite of the absence of numerous immature blood forms in the circulating blood, because of the marked bone marrow activity found at autopsy, and he believed that in Susstrunk's case the hematopoietic system was somewhat more fully developed than in his own case. The fatal case reported by Frank showed practically no immature blood cells at the time of the first examination, but subsequently an increasing number of both myeloblasts and erythroblasts were found until death occurred at twelve days. In the case reported by Pasachoff the anemia on the fifth day was the most severe yet reported (R. B. C. 400,000: Hg. 8 per cent), with only an occasional normoblast in the circulating blood. The examination of the bone marrow, liver and spleen revealed an almost total absence of erythrogenic activity.

Numerous observers have reported the occurrence of erythroblastosis in "hydrops universalis congenitus," and more recently attention has

been called to a similar finding in cases of icterus gravis, particularly in the familial or "habitual" type. Although most authorities assume the presence of a toxic process in the cases of icterus gravis, histopathologic studies usually show no evidence of infection but suggest a persistence of the activity of the various organs concerned in fetal hematopoiesis. The cases reported by Kleinschmidt[7] and Buhrman and Sanford[8] are of this type. Strikingly similar pathologic findings are reported by Ferguson[9] in his six cases of "erythroblastosis in the newly born," two of which showed marked generalized edema at birth, three severe jaundice, and the remaining case neither jaundice nor edema.

CONCLUSION

Three cases of anemia in the newborn with recovery are reported. They were successive babies of the same parents. The anemia in these cases was apparently due to a temporarily inadequate erythropoiesis, dependent upon some unknown factor.

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XANTHOMATOSIS (LIPOID HISTIOCYTOSIS)

REPORT OF A CASE

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THIS condition was first described by Christian as "Defects in the Membranous Bones, Exophthalmus and Diabetes Insipidus: An Unusual Syndrome of Dyspituitarism." Since that time numerous other observers have reported the condition, making forty-five cases available in the literature, up to the present time. Rowland was the first observer definitely to classify it as an entity and to discover the metabolic defect.

Xanthomatosis is a disease of lipoid metabolism, with storage tumors of lipoid appearing in various organs and tissues. Under this heading may also be classified (1) Gaucher's disease, (2) Niemann-Pick's disease, (3) diabetic xanthoma, and (4) the xanthomas of icterus and pregnancy. In Gaucher's disease (splenomegaly) there is a tendency for occurrence in more than one member of the family, commencing in childhood, with a progressive enlargement of the spleen, leucopenia, a tendency to hemorrhages and a brownish discoloration of the skin. The typical Gaucher's cell is believed to be derived from the reticuloendothelial system and to owe its peculiar characteristics to the deposition of lipoid substance in the protoplasm. Niemann-Picks disease or hepatosplenomegaly is racial, occurring in Jewish infants, is allied to Gaucher's disease, appears in early infancy invariably before the age of two years, and involves the spleen, liver, thymus, adrenals and connective tissue, and is fatal.

Schüller-Christian's disease is characterized by involvement of the bones of the skull and occasionally other bones, with frequent association of diabetes insipidus, exophthalmus, gingivitis, and occasionally by dwarfism or adiposogenital dystrophy.

The etiologic factor common to all these conditions is a disturbance in the lipoid metabolism with a resulting deposit of lipoid substance in the organs and tissues due to the phagocytic action of the reticuloendothelial system, which either removes an excess of lipoids from the tissues, or stores an excess in certain areas; trauma or infection having stimulated a collection of histiocytes.

Chemically, the lipoids are kerasin (Gaucher's disease), lecithin (Niemann-Picks disease), cholesterol in Schüller-Christian's syndrome, and cholesterol or its esters in the essential xanthomatoses. Work is being

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done on other lipoids, and Amaurotic family idiocy (Tay-Sachs) may soon be classed as a lipoid phenomenon.

All of the reported cases present almost identical clinical and anatomic pictures. The principal feature is multiple cranial defects of varying sizes and shapes, involving both tables of the skull as well as the base, particularly in the region of the sella turcica and supra orbital plates. Often the bones of the pelvis, spine and ribs are involved. There is an associated exophthalmus which may be extreme. Swollen gums and loose teeth are not present in all cases. Diabetes insipidus is of frequent occurrence, and there may be involvement of the liver, spleen and lymph glands. If the lungs are involved, as occurs in severe cases, there may be marked dyspnea.

Roentgen-ray studies reveal extensive osseous defects of the bones of the skull and in some cases defects of the flat bones of the pelvis, spine, scapula and ribs. There is a normal stage of ossification and development of the other bones of the body. The defects in the skull cause an appearance which can best be described by comparing them to the irregular holes in a bit of moth-eaten flannel or the appearance of a pasteboard box gnawed full of holes by mice. While all the symptoms and signs described are characteristic of Schüller-Christian's disease, no one sign or symptom is essential to the diagnosis. Roentgen-ray examination of the bones depending on the symptoms or signs reveals the lesion.

It has been found that the disease is liable to spontaneous remissions. In Rowland's case there was an improvement on a low fat diet, but thyroid and pituitary extracts were given in addition. One case of Sosman's showed benefit on a low fat diet, but two other of his cases showed increased activity while on this diet. A low fat diet is logical with the excessive blood cholesterol. Pituitary and thyroid extracts have been used with varying success in the treatment, the first in controlling the diabetes insipidus and the latter for its effect on the bones. Parathyroid, irradiated foods, high calcium diet, cod liver oil, heliotherapy and insulin in the severe malnourished cases, have all been used. Roentgen-ray therapy has given the most beneficial results in Sosman's cases, and others have reported cases so treated. This is given in small, suberythema doses, the irradiation acting on the fat-laden histiocytes liberating the lipoids and allowing the lesion to heal normally.

A number of cases of this syndrome have been reported under various names, and a great many true cases have probably been missed. Rowland first suggested the term "xanthomatosis," but Wolbach suggests the term "lipoid histiocytosis" as more descriptive. However, Schüller-Christian's disease is the one accepted or "lipoidosis, Schüller-Christian type."

CASE REPORT

A boy, three years of age, was first admitted to the Children's Hospital, on January 13, 1929, with chief complaint of swelling above the right eye. His present illness began the previous June, when the child fell, hurting his right arm and leg. He was taken to a hospital at that time, where the parents were assured there were no fractures. Shortly after that the parents noticed a swelling above the right eye, not tender or painful. This swelling remained a few days and then disappeared, but had recurred at frequent intervals since that time, never completely disappearing. Before the swelling enlarged, the right eye bulged out, this being particularly noticeable when he was asleep. Each time prior to the swelling the child was cross and irritable.

Fig. 1.—Taken at time of first admission, January, 1929.

Both parents, two brothers, aged five and twelve, and four sisters, eight, fifteen, eighteen and twenty years of age, were living and well. There was no history of tuberculosis, cancer, etc.

He was born at full term, after a normal delivery and labor lasting five hours, and weighed eight pounds. He was breast fed for fifteen months. He sat alone at six months of age and talked and walked at the usual time. There had been no impairment of health, and he had gained constantly in weight. His appetite was good, the bowels were regular, and there was no excessive thirst or polyuria. Previous illness was a mild attack of mumps.

Physical examination revealed a well-nourished and developed boy, not acutely ill. The fontanel was closed. The hair was abundant, not coarse. The scalp showed a few crusted areas. Above and lateral to the right eye there was a firm elevated swelling about the size of a fifty-cent piece, raised about one-half centimeter and

Fig. 2.—Taken January, 1930, one year later, at time of second admission.

Fig. 3.—Taken after roentgen-ray treatments were instituted, showing healing of lesions.

not tender. The pupils were equal and reacted to light and accommodation. The ears were negative. The nose was flattened giving the appearance of a saddle nose. The teeth were in good condition, but dirty. The pharynx was red and the tonsils were large. The chest was normal in contour, the lungs resonant throughout with no râles. The heart was not enlarged, the action was regular. There was a loud systolic murmur, best heard over the precordium and not transmitted to the axilla. The skin showed no eruption and was not dry. The extremities were negative, the reflexes were present.

On admission the hemoglobin was 70 per cent. The red blood cells numbered 4,400,000, the white cells 12,600. A differential count showed polymorphonuclears 71 per cent, lymphocytes 26 per cent and transitionals 3 per cent. There were no abnormalities noted in the stained smear. The Wassermann reaction was negative, and 0.1 milligram of tuberculin given intradermally, was negative in twenty-four and forty-eight hours. The urine was not increased in amount, straw colored, acid, with no albumin sugar or acetone. The reaction for the Bence-Jones body was negative. Sugar tolerance test showed a normal curve. The blood calcium was 12 milligrams per 100 cubic centimeters of blood.

Ophthalmoscopic examination showed no choking or fullness of the discs. The pupils reacted to light and accommodation. There was a slight exophthalmus measuring 2 to 4 millimeters on the right. There was no lid lag.

X-ray examination revealed multiple defects in the bones of the skull, ilium and scapula. Diagnosis from x-ray pictures was multiple myeloma.

During his stay in the hospital there was no elevation of temperature or pulse. He was discharged January 21, 1929, with a diagnosis of multiple myeloma.

He was readmitted December 26, 1929, when x-ray examination revealed much more destruction of skull without changes in other lesions. Examination at that time revealed numerous soft swellings with depressions over skull, greatest over the left frontal area. The liver was enlarged and palpable one and one-half finger-breadths below the costal margin. The blood cholesterol measured 142 milligrams per 100 centimeters of blood. He was discharged on January 4, 1930, with a diagnosis of xanthomatosis.

After discharge from the hospital, he was followed in the out-patient department, where roentgen-ray therapy to the involved bone areas was given. There was some improvement. Unfortunately the patient moved to another city, and later reports were to the effect that the disease had progressed with the appearance of a severe diabetes insipidus.

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CONGENITAL MALFORMATION OF THE BILE DUCTS

A Report of Three Cases in One Family

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CONGENITAL atresia of the bile ducts was recognized and described as a pathologic entity early in the latter part of the eighteenth century. In 1891, John Thomson[1] collected forty-nine cases from the literature and added one case of his own to give the first complete and correlated description of the disorder.

Thomson was quite impressed with the familial incidence of the malformations and in his review cited the cases reported by Gould,[2] Bing,[3] Glaister,[4] and Thomas.[5] Gould in 1855 reported a family in which the first pregnancy resulted in the birth of a premature infant who died at the age of sixteen days of hydrocephalus and congenital ichthyosis. The second, third and fourth pregnancies were terminated by the birth of normal infants. The fifth child, a male, had a roughened hard skin at birth, shortly became jaundiced and passed clay-colored stools and dark urine. He died on the twelfth day of umbilical hemorrhage and at necropsy was found to have impervious cystic and common ducts. The sixth child, a female, likewise had a roughened, hard skin. She passed one meconium stool after which the motions were clay-colored. She became jaundiced on the sixth day and died from umbilical hemorrhage on the eighth day. At necropsy, the cystic and hepatic ducts were closed, though the common duct was patent. Bing also reported two proved cases in one family, and Glaister reported a case occurring in the eighth pregnancy of parents of whom four previous children had died after almost identical illnesses. Thomas reported another case in an infant of whose siblings four had died previously with exactly the same symptoms.

Among the more recent writers the reports of familial incidence were so infrequent that Holmes[6] in his classical analysis merely mentions the fact that they do occur. Dunbar,[7] however, reported an instance of the condition in the last member of a family of eleven children, four of whom reached adult life and six of whom died in early infancy following deep jaundice and clay-colored stools. The reported patient maintained an "uneasy" existence for seven months. Jaundice had been noted at the time of birth. The stools were hard and scanty and were streaked but were not admixed with yellowish brown coloring matter. At necropsy, "the bile ducts seemed to be replaced by mere

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connective tissue." Gordon[8] from this clinic reported the case of a child whose older brother had become markedly jaundiced on the second day and died on the fifth day of life. The reported patient had an almost identical history with jaundice after the second day, dark urine, and clay-colored stools. The patient died on the sixth day of life from umbilical hemorrhage and was found to have complete occlusion of the common bile duct. Among the twenty proved and ten presumptive cases of congenital obliteration of the bile ducts seen in this clinic, exclusive of the present series, Gordon's case is the only one in which there is even a suspicious history of familial incidence of the malady.

The rarity of the familial incidence of congenital atresia of the bile ducts makes the following series of three cases occurring in one family worthy of publication. It is of interest to note that in two of these infants there was an associated congenital anomaly of the heart.

The family history of these patients gave no evidence of syphilis or other significant disorders. The sequence of pregnancies was as follows:

I. A miscarriage. The date and the age of the fetus are not remembered.

II. A female white infant who died at the age of one year and nine days as a result of a congenital malformation of the bile ducts. (See Case 1.)

III. A female child who apparently was well at the age of five years.

IV. A male infant who died at the age of fifty days as a result of a congenital malformation of the bile ducts. (See Case 2.)

V. A male infant who died at the age of forty days as a result of a congenital malformation of the bile ducts. (See Case 3.)

CASE 1.—E. W., was a female infant who was born at term by normal delivery on March 17, 1924. She was admitted to the Surgical Service of the Children's Hospital on March 14, 1925, at the age of one year. She had had no symptoms save those directly associated with the presenting illness. She had been jaundiced from birth, the jaundice at times being more pronounced than at others with some periods of remission. She slept poorly but had a normal appetite and did not vomit. The stools were almost white with occasionally a yellow tinge. The urine was very dark. Pruritis was said to have been severe.

The patient was a well developed and very well nourished female infant with deep jaundice of the skin and sclerae. The heart was not enlarged to percussion but on auscultation there was a soft systolic murmur over the whole anterior chest. This was transmitted through to the scapular region. The abdomen was not enlarged. The superficial veins over the epigastrium were dilated and prominent. The liver was palpable one inch below the costal margin. The spleen was not felt. The urine showed a trace of albumin and a large amount of bile. The stools were acholic. There was a moderate secondary anemia. The coagulation time of the blood was slightly prolonged.

A diagnosis of congenital atresia of the bile ducts was made. While awaiting exploratory operation, the patient developed an acute respiratory infection with a temperature of 104°. Two days later she began to pass blood in her stools. She

had diarrhea with from 15 to 20 stools a day, each stool consisting of small flecks of fecal material and a large amount of dark blood. Her spleen became enlarged and easily palpable. The findings were otherwise normal. She responded to treatment for a short time, then collapsed suddenly and died on March 25, 1925, at the age of one year and nine days.

Necropsy.—The gastrointestinal tract was filled with blood and mucus. The spleen and mesenteric lymph glands were enlarged, soft and edematous. The liver was normal in size and of a mottled green, yellow, and red color. The parenchyma was soft with areas of firm connective tissue scattered throughout. The gall bladder was small and contained but 2 or 3 c.c. of greenish-yellow fluid. There was a very small duct extending from the gall bladder to the duodenum, and there were a few tiny ducts running from the liver edge to the gall bladder. There were two fibrous cords between the gall bladder and the hilus of the liver. No patent hepatic ducts could be demonstrated.

Microscopic examination of the liver showed varying amounts of bile pigment within the cytoplasm of the liver cells. There was also moderate periportal necrosis with early fibrosis. The intrahepatic ducts were very scarce and in places were represented by solid cords of epithelial cells. Some were patent. The finer bile canalieuli could be demonstrated easily, but in no place could they be found to contain any bile.

The right ventricle of the heart was uniformly enlarged but there was no other abnormality.

Diagnosis.—Congenital malformation of the bile ducts with atresia and partial absence of the hepatic ducts.

Congenital malformation of the heart with idiopathic hypertrophy of the right ventricle.

CASE 2.—R. W., a male infant, was born at term by normal delivery on May 13, 1929. He was admitted to the Infants' Hospital on June 21, 1929, at the age of five weeks. He was slow in regaining his birth weight and on the tenth day had a severe attack of cyanosis. Examination at that time showed an enlarged heart, which was confirmed by x-ray examination, and a rough systolic murmur. On the third day of life jaundice appeared. This gradually deepened and by the third week was quite intense. The stools were clay-colored, the urine was dark. Pruritis was said to have been marked. There were no other symptoms.

The patient was a fairly well developed and well nourished white male infant, deeply jaundiced. The heart was slightly enlarged, and there was a rough systolic murmur heard over the entire precordia. The liver and spleen were palpable. The findings were otherwise normal. The blood showed a slight leucocytic increase. The bleeding time was four minutes, the coagulation time twelve minutes (capillary tube method). The icteric index was 100 to 150 units. The fragility of the erythrocytes was slightly decreased, hemolysis beginning in 0.40 per cent and being complete in 0.28 per cent NaCl. The urine contained a large amount of bile. The stools were acholic and contained a large amount of fat. The Wassermann reaction and the Mantoux test were negative. Roentgenograms showed that the heart was enlarged to the left; films of the chest, abdomen, and long bones were negative.

A diagnosis of congenital atresia of the bile ducts was made and on the fifteenth day in the hospital, July 6, 1929, an exploratory laparotomy was performed. The liver was large, soft, dark, and plum-colored. There was a small amount of yellow, bile-stained fluid free in the peritoneal cavity. The gall bladder appeared to be rudimentary. The common and cystic ducts were represented by small fibrous cords, and there was no evidence of a patent hepatic duct. There appeared to be no hope of surgical repair. The patient gradually failed and died on the twenty-sixth day in the hospital at the age of fifty days.

Necropsy.—The liver was normal in size and was a greenish-brown in color. The gall bladder was spherical in shape and was distended with mucoid, colorless material, which was expressed through the ampulla of Vater by exerting considerable pressure on the gall bladder. The hepatic duct was represented by a fibrous cord. There was a marked constriction of the neck of the gall bladder and the cystic duct. The spleen was not abnormal.

Microscopic sections of the liver showed some regions of very widespread necrosis with early fibrosis. In other areas the liver architecture was essentially intact. The intrahepatic bile ducts were few in number. All were free from bile. Most of them exhibited very small lumina, and frequently they were represented by solid cords of epithelial cells. The liver cells and phagocytic cells contained varying amounts of bile pigment. This was principally within the cytoplasm of the cells, and though the finer bile canaliculi were readily demonstrable, there was no evidence that the liver cells had extruded bile into any of them.

The heart was enlarged to the right with a patent ductus arteriosus. There was an interventricular septal defect, and the free borders of the mitral and tricuspid valves were thickened slightly.

Diagnosis.—Congenital malformation of the bile ducts with atresia and partial absence of the hepatic ducts. Congenital malformation of the heart with interventricular septal defect.

Case 3.—B. B. W., a male infant, was born at term by normal delivery on January 2, 1931. He was admitted to the Infants' Hospital on January 16, 1931, at the age of two weeks. He had been under observation at the Boston Lying-In Hospital during his first two weeks of life. At the age of one week, the patient became jaundiced. Three days later it was noted that he was passing clay-colored or only slightly green-stained stools which contained a large amount of fat but no bile. He was not vigorous and after his initial loss made no substantial gain in weight.

The patient was a moderately well developed but poorly nourished white male infant who was deeply jaundiced. The liver was palpable two fingerbreadths below the costal margin. The spleen was not felt. The findings were otherwise normal. The erythrocytes, the leucocytes, the bleeding time, and the coagulation time were normal. The icteric index was 150 to 200 units. The fragility of the erythrocytes was slightly decreased, hemolysis beginning in 0.44 per cent and being completed in 0.18 per cent NaCl. The urine contained a large amount of bile. The stools were acholic and contained a large amount of fat. The Wassermann reaction and the Mantoux test were negative. Roentgenograms of the chest, abdomen, and extremities were essentially normal.

A diagnosis of congenital atresia of the bile ducts was made, and an exploratory laparotomy was performed on the fifth day in the hospital, January 21, 1931. The gall bladder was small but contained a viscous fluid. The cystic and common ducts appeared to be small and obliterated, but, when pressure was exerted on a catheter introduced into the gall bladder, the ducts seemed to dilate. As the procedure had been successful in other cases in this clinic, the catheter was left in and an attempt was made to dilate the ducts by gentle pressure. This was not successful. The patient had considerable postoperative reaction with a fluctuating temperature and frequent vomiting. He gradually failed and died three weeks after operation at the age of forty days.

Necropsy showed fibrinous adhesions at the site of operation between the anterior abdominal wall, the liver, and the intestines. The gall bladder was small. Dye introduced into the cavity under pressure escaped readily through the ampulla of Vater into the duodenum. The common duct was tied, and dye again was introduced into the gall bladder under pressure. This could be followed up the

hepatic ducts to the point of bifurcation in the hilus of the liver. Beyond this point the hepatic ducts were of very small caliber. The liver was slightly larger than normal. It was of a mottled green color and on gross examination appeared to be somewhat fibrosed. The spleen was essentially normal.

In microscopic section, the liver showed a moderate amount of cirrhosis which was most evident around the portal spaces. The liver trabeculae in general were well defined. There were dark staining granules of bile salts in many of the liver cells; about some cells in which this was most marked there were focal areas of necrosis. There was a slight but definite increase in the number of mononuclear phagocytic cells and polymorphonuclear neutrophiles throughout the liver. The intrahepatic bile capillaries were normal in number and in distribution. The finer canaliculi were readily demonstrable, though there was no evidence of excretion of bile into any of them. In the hilus of the liver the bile ducts were patent though very small. They were followed downward by serial sections and at a point just above the bifurcation of the hepatic ducts were shown to consist of solid cords of epithelial cells. Beyond this point they were again patent.

The heart was normal in size and without anatomic defect.

Diagnosis.—Congenital malformation of the bile ducts with atresia of the hepatic ducts.

COMMENT

The etiology and pathology of this condition have been discussed thoroughly by Thomson,[1] Rolleston and Hayne,[9] Howard and Wolbach,[10] Holmes,[6] and many others. Ladd[11] has described the surgical treatment. Without attempting a complete discussion of these subjects, there are a few outstanding features in this series of cases which deserve mention. In all three cases the lesions were essentially the same, and in two of the cases there were associated congenital malformations of the heart. In the same two cases the smaller bile ducts were much less numerous than they are in a normal liver. This may be taken to indicate a definite absence of part of these ducts. All three cases showed a complete absence of excreted bile in the finer bile ducts and canaliculi. This last fact makes it seem probable that the disorder was not confined to the bile ducts alone but was either coincident with or superimposed upon a basic physiologic dysfunction of the liver cells themselves. The liver cells, in other words, while anatomically normal were physiologically imperfect and apparently incapable of excreting bile.

The combination of three almost identical cases in one family, the association of other congenital abnormalities in two of the cases and the replacement of part of the bile capillaries by solid cords of epithelial cells in all three cases, point rather definitely, in this series of cases at least, to a congenital malformation or maldevelopment rather than an intrauterine or other infection as the etiologic agent. The same considerations also bring strongly to the fore the question as to just how much significance should be placed on the rôle of heredity in these patients.

The fact that one of these patients lived in a state of good nutrition and development for over a year with complete atresia of the bile ducts is especially worthy of attention.

SUMMARY

Three cases of congenital malformation of the bile ducts with complete atresia, occurring in a single family are reported with brief operative notes on two and necropsy findings on all. Six similar cases with familial incidence of the anomaly are listed from the literature.

I wish to acknowledge the kindness of Dr. W. E. Ladd and Dr. S. B. Wolbach in permitting this report of the above cases.

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ANTIQUES OF PEDIATRIC INTEREST

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AMONG the articles of pediatric interest fashioned from pewter are included pap boats, feeding pots, bleeding bowls, castor oil spoons, croup kettles and porringers. Specimens of some of these will be illustrated later in this series.

Pewter is an alloy of tin with copper, antimony or lead in various proportions. Good pewter contains over 80 per cent of tin. Pewter utensils were manufactured by casting, by hammering a flat plate of the metal, by turning on a lathe, or by a combination of these methods.

Vessel for steeping infant's sucking bag. Pewter, German, circa 1800, capacity 3 ounces.

While pewter utensils dating from the Roman occupation have been found in England, these are excessively rare. During the eighteenth and nineteenth centuries pewter was widely used for domestic utensils. Its scarcity at the present time probably is due to its having been melted down for use as solder. Various individual trade and guild marks—known as touch marks—are frequently found on pewter but the absence of these stamps is not a sign that the article is not a genuine antique. In fact, the modern faker of old pewter is usually very careful to place a clearly impressed touch mark on his wares. In the early part of the nineteenth century Brittania metal, an alloy of tin with antimony, plus copper or zinc, was introduced. This material

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could be more easily fashioned on the lathe than pewter and the discovery that it could be electroplated spelled the doom of pewter.

The article illustrated was used for soaking the infant's comforter, or soother, in a sugar solution. Struve, in "A Familiar View of the Domestic Education of Children," English translation, London, 1802, describes the popular use of the comforter: "One of the most disgusting customs is the sucking bag, which is given to a child for the double purpose of nourishing and composing it. Many a poor mother will tear a rag from an old shirt, or a clout, which she has found perhaps in the street, and which may contain the remains of venereal contagion: of this she makes a small bag, which is filled with bread, milk and sugar, and then given the child to suck. If the infant happens to drop this rag on the ground, it is presented again, though covered with dirt: a number of flies settle upon it when the child is alone, which but the moment before may have quitted a saucer of poison. Nor are these the only bad consequences: the bread contained in the bag turns sour and the child may sometimes swallow the whole rag, and be choked by it. Let me entreat all mothers, as well as every person employed in the care of children, not to continue the use of this bag longer than the first eight weeks, or what is still better, to give it only at the time of drinking; as many nurses imagine that they should not administer any drink without using this bag, lest some of the liquid pass into the trachea, or windpipe. It is absolutely necessary to wash the bag, and not suffer particles of bread, biscuit, roll, &c. to remain in it too long; it ought to be filled with fresh food every time it is used. Finally, this bag should not be steeped too often; for, with the liquid, the child imbibes a quantity of air, which occasions flatulence and gripes."

Critical Review

TUBERCULOSIS IN CHILDREN

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A REVIEW of recent literature upon tuberculosis in children under 15 years of age with the aim in mind of portraying present-day concepts of the problem, necessarily requires considerable change in many ideas which were orthodox even as short a time as a decade ago, and, indeed, which are still contained in our most modern textbooks.

While many of the facts were known, yet they had not been correlated into an understandable whole, and the attack upon tuberculosis, as the worst disease of mankind, was directed chiefly toward campaigns for early diagnosis and development of sanatoriums for the treatment and isolation of the clinically diseased. Fruitful as these measures were, and as proud as the medical profession has a right to be of one of the greatest life-saving battles ever waged against a disease, yet to abolish completely the disease by these means alone was pretty nearly an impossibility. A tremendous new enthusiasm and a much brighter outlook has arisen since it has been realized that in children there lies the possibility of recognition of tuberculous infection almost from the time of its inception. Herein lies the best opportunity for the prevention of destructive disease and the dissemination of bacilli, both attributes of the adult form of tuberculosis.

From the writings of Myers, Opie, McPhedran, Kraus, Chadwick, and numerous others it has become generally understood what is meant by the term Childhood Type Tuberculosis. This term was designated by the American Sanatorium Association[1] in 1929 to describe the diffuse or nodular lesions in the lungs and associated tracheobronchial nodes that result from a first infection of the pulmonary tissue with the tubercle bacillus. It is in contrast to the adult type which is the result of a reinfection, exogenous or endogenous, implanted upon allergic soil.

Chadwick listed the essential differences between the childhood type of tuberculosis and the adult type. This list as modified by Waring[63] is as follows:

Childhood Type

1. Common in children, rare in adults.
2. Results from first infection.
3. Associated tracheobronchial lymph nodes always involved, not always demonstrable.
4. Lesion involves any part of lung, no preferred sites.
5. Caseous lesions usually followed by calcification and slight fibrosis.
6. Infiltrated areas commonly resolve leaving little or no scars except for foci caseation, which usually become calcified.
7. In general, a greater tendency to rapid spread of lesions and to development of miliary tuberculosis.

8. Cavities not apical, not close to pleura, form quickly and are more easily healed.
9. Prognosis is good.

Adult Type

1. Common in adults, rare in children.
2. Results from reinfection.
3. Tracheobronchial lymph nodes not involved.
4. Apical localization, often in lung opposite to that containing calcified primary focus and calcified tracheobronchial glands.
5. Caseous lesions usually followed by excavation and much fibrosis.
6. Infiltrated areas heal with much fibrosis.
7. In general, a greater tendency to encapsulation and to healing of lesions.
8. Cavities apical, close to, and adherent to pleura, relatively rigid and do not heal easily.
9. When this type affects a child, the prognosis is bad.

McPhedran[37] in a splendid presentation has described in detail the parenchymal and tracheobronchial lymph node lesions characteristic of childhood tuberculosis based upon roentgenologic evidence. A chart is appended to his article with illustrations of the various lesions and also a synopsis indicating the salient features of each lesion, such as descriptive term, x-ray appearance, tuberculin reaction, physical signs, weight, fever, symptoms, commonly associated lesions, and diagrams of the latter.

Of the parenchymal lesions the primary nodule, caseous or calcified, is the most common. When calcified it shows clearly in the x-ray film, and is frequently referred to as a Gohn tubercle. The lesion is subpleural, more often seen in lower lobes and may be obscured by the heart, diaphragm, or overlying ribs. In association with calcification of the regional lymph nodes it is the most common tuberculous lesion seen in children between the ages of four and puberty and is known as the "primary complex of Ranke." Its chief significance is in indicating a previous tuberculous infection. In x-ray films the lesion can easily be confused with the round dots produced by blood vessels being situated axially to the film.

Consolidations and diffuse infiltrations of the lung may occur which are either progressive and serious, or retrogressive and benign. Serial plates are frequently the only means of determining which way the lesion is tending. Cavitation may occur or the lesion may clear in the course of weeks or months leaving a calcified area or a few strands. Such extensive lesions as these are more likely to be seen in infants, whose exposure to contagion is intimate and prolonged, and, occasionally, in older children, particularly colored. Miliary tuberculosis, frequently associated with tuberculous meningitis, is the common terminal lesion of this age period. It results when bacilli escape beyond the confines of the glands or local parenchymal lesion either by way of the blood stream or from rupture of a caseous focus into a bronchus.

Tuberculous tracheobronchial lymph nodes may be uncalcified and caseous, partially calcified and caseous, or entirely calcified. Caseous lymph nodes are always serious because they frequently are the precursors of the fatal bronchopneumonic or miliary forms of the disease. Their recognition is important and difficult. Physical signs are lacking. Symptoms are not pathognomonic. "The only way to arrive at a

proper diagnosis," conclude Armand-Delille and Lestocquoy,[2] "is to supervise all children who have been in frequent and close contact with a germ carrier, and to have good roentgenograms (frontal and lateral) carefully examined, comparing them with films from other cases in which roentgenographic observations have been checked at autopsy." If calcification has occurred in the nodes they are readily revealed on the x-ray film, but caseous nodes do not show on the x-ray films unless massive enough to cast a shadow beyond the normal structures of the mediastinum.

In addition to these pathologic lesions of the childhood type of tuberculosis, Myers,[44] from observation at the Lymanhurst School for Tuberculous Children, and Opie,[47] from similiar observations at the Henry Phipps Institute in Philadelphia, have drawn attention to very significant apical lesions (adult type) which they begin to see around the age of twelve and with increasing frequency through the teen ages. By serial roentgenology they have watched these lesions through a latent period of several years before symptoms and physical signs appeared. In over half of these cases they have been able to demonstrate in the x-ray films evidence of a previous childhood type of tuberculosis. Concerning these cases Rathbun[53] says, "In our high school and junior high school students one-half of the cases of pulmonary tuberculosis have signs of latent childhood tuberculosis. This type (childhood tuberculosis) is found in only between three and four per cent of the total school population of our country, which means that 50 per cent of the cases of pulmonary tuberculosis developing during the teens is confined to this small group. I believe that 75 per cent of the potential cases of pulmonary tuberculosis that will develop the disease during or just before the teens are included in a group of children comprising those with childhood tuberculosis, their brothers and sisters, and other known contacts without demonstrable signs of disease." Such observations and conclusions rather revolutionize some of our previous conceptions. At least three deductions can be made. It would seem that it is not as desirable for healthy children to react positively to tuberculin as we once generally believed. Second, that much of the adult type of tuberculosis occurring during the teen ages and which is accompanied by such a high mortality rate is going to occur in a group of children who have come from a tuberculosis environment, and that therefore tremendous importance attaches itself to a detection of and a breaking up of this situation. Third, that our previous ideas of what constituted early diagnosis of the adult type of tuberculosis by means of stethoscope, physical signs, and symptoms must be replaced in favor of information gained by tuberculin testing and x-ray evidence.

Abundant evidence has accumulated in numerous studies in recent years to prove beyond question that the diagnosis of such tuberculous lesions as I have just indicated cannot in the majority of instances, be made on physical findings or symptoms, for both these frequently, and as a rule, are lacking, or, if present, are more likely to be due to some other condition. Signs once thought to be of value in the diagnosis of lymph node involvement such as D'Espines sign, Eustace Smith sign, interscapular dullness, or dullness over the sternum have been reported by many careful observers to be of little value. Extensive parenchymal lesions of the retrogressive and benign type are noted for their paucity of physical findings, quite at variance with what would be expected from the appearance of the x-ray film. Hetherington and others have

repeatedly shown that the undernourished child as judged by weight is no more likely to be tuberculous than are his normal or overweight associates. Sputum examinations are of little help because of the habit infants and children have of swallowing their sputum. Poulsen[51, 52] reports some success in demonstrating tubercle bacilli in the stomach contents by the following method: In the morning after a fast of 6 hours the stomach is washed with 200 to 300 c.c. of sterile water; the wash water is centrifugated; sediment homogenized, and stained with Ziehl-Neelson's method. If negative, cultures from the sediment are made on Petroff's medium, and guinea pigs are inoculated. Such a procedure, however, will probably not find any very widespread application because of the technical difficulties involved.

I do not mean to give the impression that careful physical examination, proper evaluation of symptoms, and search for tubercle bacilli, are measures that can be ignored in diagnosis or study of progress of the lesions. Dullness and bronchial breathing over pulmonary lesions, râles, fever, cough, and fatigue are all indicative of manifest disease and are encountered frequently enough to require that this phase receive careful consideration. But the point to be emphasized is that very significant tuberculous lesions of the childhood type and of the adult type exist in children in a latent form which are not accompanied by physical signs or by symptoms.

Chief dependence for a diagnosis of tuberculosis in children must be placed upon history of exposure, the tuberculin reaction, and roentgenology.

Tuberculosis is a definitely contagious disease and any child who has been in contact with a germ-positive case is likely to have become infected, and the more intimate and prolonged the exposure the more certainly that infection will have taken place. Contact, of course, frequently occurs unknown, but in general it may be said that infection occurs four times as frequently in the contact group as in the noncontact group. Infants in a tuberculous household have little or no chance of escaping infection. A certain amount of tuberculous infection is of bovine origin acquired through the drinking of infected nonpasteurized milk but compared to the danger from human source is relatively insignificant.

In the tuberculin test we have a definite and proved means of detecting tuberculous infection. Krause[34] has shown that the first tissue response to an invasion by tubercle bacilli is a proliferative one—the formation of anatomical tubercle. Within two weeks (more or less) after the formation of anatomical tubercle the tissues have taken on a new type of reaction, exudative in character, which is given the term hypersensitivity or allergy. All the tissues of the body develop this property and an allergic or exudative response results whenever and wherever tuberculo-protein is introduced into the tissues.

Most pediatricians are familiar with tuberculin testing and employ it routinely in their practices. On the other hand most general practitioners are not familiar with it and do not use it in their practices, and since the majority of the children of the nation are dependent upon the family physician the desirability of greater use of tuberculin testing by this group of physicians is apparent. Experience has shown that the Mantoux or intradermal test is more reliable than any other type of tuberculin test. It should, therefore, be used exclusively in order that

the comparing of results may be more accurate. Most authorities agree that 0.1 c.c. of 1:1000 dilution giving 0.1 mg. is a safe first dose. This will permit the picking up of the great majority of tuberculous conditions. It is customary to repeat the test, if the first one is negative, using 1.0 mg. before assuming that tuberculous infection is absent. This is desirable and probably should be done in hospitals, dispensaries, etc., but in private practice where the relation of patient to physician is much more intimate it is much better to get one done than none at all.

The objections that have been raised to the Mantoux test are that it has to be done with a needle and that dilutions have to be made frequently. The former is hardly valid, since most people are accustomed to having needles used in the giving of diphtheria inoculations or in Schick testing, and rarely is an objection encountered if a careful explanation is given that it is merely a local test for possible exposure to tuberculosis. As to the latter objection Smith suggests an arrangement with the druggist for those physicians not arithmetically minded. In Iowa the problem has been very satisfactorily solved through the courtesy of the State Tuberculosis office in offering to send out to any physician in the state, tuberculin, properly diluted, at intervals sufficiently frequent to insure a potent material.

In performing the test it is important that all the material go into the skin. If done correctly a wheal is raised in which can be seen the individual hair follicles. Readings are made at the end of 48 hours. Edema is more significant than redness as a measure of hypersensitivity. Opie and McPhedran have suggested the following method of interpreting results: The intensity of the reaction has been recorded as one plus (+) when redness and edema are present in an area of 10 mm. in diameter. Reactions with redness and edema in an area from 10 to 15 mm. in diameter have been designated two plus (++). Extensive redness and edema measuring more than 15 mm. have been recorded as three plus (+++) or, if accompanied by necrosis of the skin as four plus (++++).

The allergic response is absent in the pre-allergic period—after infection occurs and before allergy develops; may be absent or depressed during the acute communicable diseases such as measles, scarlet fever, diphtheria, etc.; may be absent when the tuberculous lesion has become completely healed so that no tuberculo-protein reaches the circulation; and may be absent or depressed when overwhelming tuberculous disease is present. With these exceptions the tuberculin test can be relied upon to be at least 90 per cent efficient. It should be kept in mind that the tuberculin reaction is a test for skin hypersensitivity or allergy, and is not a test for activity of the infection, nor does it give any information as to the amount of disease present.

Mariette and Fenger[36] have reported their findings in the use of M A 100, a purified tuberculo-protein substance, in 4000 cases in comparison with Old Tuberculin. They found M A 100 equal to and slightly superior to Old Tuberculin as a testing agent and recommend the use of the latter in the interest of uniform results.

Every child who reacts positively to the tuberculin test should have an x-ray of the chest and if necessary oblique and lateral views. X-ray is an essential in diagnosing the extent of the diseased process, and in directing treatment. If the chest is negative it should be remembered that the primary focus may be in the abdominal cavity or elsewhere.

Dunham and Smythe[19] were able to show calcified mesenteric lymph nodes in x-ray films. Calcified cervical glands have also been shown roentgenologically.

Children with negative films but with positive tuberculin tests should have x-ray films of the chest repeated at least every six months. Children at the age of puberty and beyond who are known to have a childhood type of infection need careful watching roentgenologically for the beginning of a latent adult type of tuberculosis.

Numerous surveys have been undertaken in this country in recent years designed to show the amount of tuberculous infection existent particularly among school children. The findings have been quite at variance with what we formerly believed to be universally true of all children based upon figures of Hamburger and Monti[26] obtained in 1909 in Vienna. These authors reported that of children five years of age approximately 51 per cent reacted, of children ten years of age 72 per cent, and of children fifteen years of age slightly more than 94 per cent. They used the intracutaneous test. Veeder and Johnston[62] in St. Louis in 1915 were the first (in this country) to draw attention to the fact that at least infection was not universal. In 1321 children they found, using the von Pirquet test, only 48 per cent positive at 13 years. Among the lowest figures to be obtained are those of Slater[56] who in 1924 in a rich farming community in Minnesota among 1654 school children found by the von Pirquet method only 10 per cent infected. Slater and Jordan[57] have recently repeated this work using both the Mantoux and von Pirquet methods of testing. Of 1006 children they found 13.3 per cent positive. This is in striking contrast to the figures obtained by Hetherington, McPhedran, Landis, and Opie[29] in certain schools in Philadelphia. These authors using the intradermal test in three strengths 0.01 mg., 0.1 mg., and 1.0 mg. found among 4107 children 38 per cent infected at 5 years of age; 71 per cent at 10 years of age; and 81 per cent at 15 years of age. Korns,[31] using the same method of testing, reports that only 10.2 per cent of 1103 healthy school children of Cattaraugus County, N. Y., reacted positively. Myers found 50.4 per cent positive reactors of 6000 children tested at the Lymanhurst School for Tuberculous Children. Smith[58] reports the results of Mantoux testing in the children's division of the Bellevue Hospital from 1921 to 1928. The study comprises tests done on 7668 children of whom 1286 were found positive. By age groups the first six months showed 3.38 per cent positive reactors; from 6 months to 2 years, from 8 to 12.5 per cent; from 2 to 6 years 15.6 to 18 per cent; from 6 to 10, 26.3 per cent to 29.7 per cent; from 10 to 13 years, 38.8 per cent to 43 per cent.

The most extensive program of tuberculin testing has been carried on among school children in Massachussetts. In the first 100,000 children tested by the von Pirquet method approximately 28 per cent have given positive reactions; of 42,000 more recently tested 20 per cent reacted.

Such figures as the above show considerable variation in the amount of tuberculous infection found among children. This is to be explained upon the type of test used, whether von Pirquet or Mantoux, and upon the type of community in which the testing was done. It would seem desirable for purposes of accuracy in comparing statistics that a uniform method of performing tuberculin tests be adopted. It must be admitted, however, that there is considerable evidence to show that the

majority of significant tuberculous lesions in the chests of children will be revealed by the von Pirquet test or by the smaller dosage (0.01 mg.) by the Mantoux method (Pope, Hetherington, et al., Slater and Jordan).

The results of roentgenologic examination of children's chests who react positively to tuberculin have been recorded in several investigations. Hetherington and his associates in their study of school children in Philadelphia x-rayed 467 children between the ages of 10 and 14, and 1066 between the ages of 15 and 19. Pulmonary nodules were found in 13.9 per cent of the former group and in 20.1 per cent of the latter group; latent infiltration of the lungs (childhood type) in 2.1 per cent of the former and 1.9 per cent of the latter; latent apical tuberculosis in 0.5 per cent of the former and 1.0 per cent of the latter; manifest tuberculosis in 0.6 per cent of the former and 0.7 per cent of the latter.

Barnard, Amberson, and Loew,[4] in a study of tuberculosis in 1000 adolescent children in New York City, tested to tuberculin and x-rayed the entire number. Six hundred seventy-three gave a positive tuberculin reaction; in 184 there were tuberculous lesions as demonstrable by the x-ray; 171 of these were positive tuberculin reactors and 13 were negative; 71 were classed as apparently healed and 113 as clinically important. In 26 cases evidence of the disease was found in the pulmonary parenchyma only; in 89 in the tracheobronchial region (lymph nodes) only; and in 69 in both situations. The summary of their x-ray observations is particularly interesting.

"If we had x-rayed only the 673 who had a positive reaction to the tuberculin test, we would have discovered 173 of the 184 with tuberculous pulmonary lesions demonstrable by x-ray—all but eleven.

"If we had x-rayed only the 168 who were graded D on general health, we would have discovered 25 of the 184.

"If we had x-rayed only the 128 who were 10 per cent or more below average weight for age and height, we would have discovered only 18 of the 184.

"If we had x-rayed only the 35 of the entire 100 who, on rather more careful inquiry than could usually be assumed were found to be exposed, or to have been exposed in their homes to a case of pulmonary tuberculosis, we would have discovered 8 of the 184—one out of 23; and if we had included all with probable, as well as known, history of contact, we would have discovered 17, or one in eleven.

"If we had selected for x-ray examination only the 24 in whom suspicious pulmonary signs were detected at the physical examination, we would not have found one."

Pope[50] and Lord[35] give the incidence of tuberculous disease discovered roentgenologically in the positive reactors in the Massachussetts survey. "Tuberculosis of the childhood type was demonstrated in 2497 (2.5 per cent) of 100,000 grade school children examined during the first five years of the Chadwick Clinics. Suspicious cases numbered 5369 and tuberculosis of the adult type was found in 98. Of the 42,022 grade school children more recently examined 400 (0.9 per cent) had the childhood type of the disease and 1060 (2.5 per cent) were classed as suspects. Thirteen (0.03 of 1 per cent) were found to have pulmonary tuberculosis.

Of 7318 high school children, evidence of pulmonary tuberculosis was demonstrated in 13 (about 0.2 of 1 per cent). This type of the disease

is thus more than six times as common in the high schools as in the grade schools. In addition, twice as many suspected pulmonary cases were found in the high schools."

Stewart[60] in an excellent paper has published the results of his observations at the Lymanhurst School for Tuberculous Children.

The report is based on a study of 5816 cases, 68.4 per cent of which were negative to the von Pirquet test and 31.6 per cent of which reacted positively. In 44.5 per cent of the positively reacting group, evidence of childhood tuberculosis was found roentgenologically, and in 3.9 per cent of those reacting negatively.

If questionable and slight calcifications are excluded the percentage rates are reduced to 25.9 in the former instance and 0.58 in the latter. Commenting on these findings Stewart says that, "If one tries to evaluate the efficiency of the tuberculin test, the roentgenogram and the stethoscope in making a diagnosis of childhood tuberculosis, and assigns an arbitrary value of 100 per cent to the tuberculin (von Pirquet) test, on this basis the roentgenogram has a value of about 25 per cent and the stethoscope (as judged by experience) a small fraction of 1 per cent."

In the 1835 children with a positive von Pirquet test Stewart found by x-ray 66 cases (3.6 per cent) of pulmonary involvement to which he gives the name "resolving parenchymal lesion of first infection." It was the only type of diffuse pulmonary lesion produced by the first infection by the tubercle bacillus that he observed in his group of positive reactors. Describing it he says, "On initial examination the roentgenologic study usually does not justify or permit an identification of the lesion as being tuberculous in origin, since simple nontuberculous pneumonic consolidation may present similar shadows on the x-ray film. However, in children with positive tuberculin tests presenting no clinical symptoms of pneumonia, the roentgenologic demonstration of pneumonic-appearing lesions which slowly resolve with the subsequent appearance of calcium deposits in the adjacent lymph nodes, with or without the development of a Gohn tubercle, a diagnosis of a resolving parenchymal tuberculous lesion of first infection is justified. In fact, after careful study a diagnosis often is justified long before calcium deposits appear. This earliest stage characteristic of the first massive infection by the tubercle bacillus constitutes a rather definite and fairly uniform clinical entity. The diffuse parenchymal lesions quite uniformly tend to recede slowly as time elapses, following which intrathoracic glandular calcifications appear. Physical signs are usually inconspicuous even in the presence of rather extensive infiltration. During the course of the resolution of the parenchymal lesion the child usually has little or no fever, often presents only moderate leucocytosis and continues to eat well and to show a satisfactory gain in weight. The deleterious effects of these lesions on the general health of the child are strikingly absent, even in instances of involvement of an entire pulmonary lobe, and stand in marked contrast with the evidence of prostration, toxicity and debility manifested by patients suffering from adult tuberculous lesions of reinfection of similar extent."

This description corresponds very closely with cases reported by Eliasberg and Neuland,[20] by Goldberg and Gasul,[24] and by Spence[59] and which have been called by them "epituberculosis." Stewart suggests the term epituberculosis may be superfluous. It is also probable that

these cases correspond to the "consolidations and diffuse infiltrations, retrogressive and benign" of McPhedran's classification. It would seem desirable that a more uniform and agreed upon classification of the pulmonary lesions of the childhood type of tuberculosis be established.

As regards calcification of intrathoracic nodes Stewart reports that for all degrees of calcification 3.4 per cent were found in the negative reacting children, and 25.7 per cent in the positive reactors. Gohn tubercles were demonstrated in 0.45 per cent of the negative reactors, and in 15.2 per cent of the positive reactors.

Adult type of tuberculosis was found in 0.075 per cent of the children who reacted negatively and in 2.5 per cent of those who reacted positively. In this group of children unmistakable evidence was discovered of the recent development of lesions of the adult type superimposed on, and post dating previously existing and nonprogressive scars of childhood or first infection tuberculous pulmonary disease. Such findings have led Stewart and Myers to regard the childhood type of tuberculosis as benign and nonfatal, and the adult as the serious and killing type. They question the opinion once held that a first infection confers protection against future disease.

One final observation on this group of children from the Lymanhurst School is worth noting and that is, that the resolving parenchymal lesions were noted most frequently in the second year of life tapering off to the eleventh year. Calcifications were noted most frequently from the fifth year on, and the adult type of disease was not noted until the sixth year of life and only occasionally from then on up to puberty when there was an abrupt rise in its incidence of occurrence.

Studies of the character just referred to have formed the basis for a considerable modification and moderation of the once serious prognostic outlook attached to a diagnosis of tuberculosis in a child. Cases of miliary tuberculosis, tuberculous meningitis, and tuberculous bronchopneumonia with positive sputum are just as fatal as they ever were, but such cases as these, in light of our present methods of recognizing latent tuberculous lesions, must be looked upon as the exceptional outcome.

Gasul[22] studied the fate of 404 tuberculous infants for a period of from one to eight years. Twenty-nine of the 404 were infected during the first six months. Of these, five, or 17.2 per cent, died; seventy-three became infected during the second half year of life of whom five, or 6.84 per cent, died. Seventy-seven became infected during the first half of the second year of life of whom five, or 6.49 per cent, died. None of the infants who were infected after the first half of the second year of life died.

Myers reported that only 6 children have died from tuberculosis out of 172 who reacted positively to tuberculin at 2 years of age or under, who were examined at the Lymanhurst School for Tuberculous Children and who have been observed for a number of years.

Undoubtedly hygienic conditions and the degree and duration of exposure to tubercle bacilli have much to do with the favorable or unfavorable outcome in the individual infant.

The frequency of Gohn tubercles and calcified tracheobronchial and hilum lymph nodes occurring in children between the ages of infancy and puberty, offers abundant evidence as to the ability of children in this age group to cope with the tubercle bacillus.

The prognostic outlook assumes serious proportions, however, for those children in their teen ages who develop the adult form of tuberculosis. In this country the mortality rate reaches its highest peak in the age group from 15 years to 25 years of age, the death rate being considerably higher for girls than for boys. According to Zacks[68] from studies in Massachussetts in children of school age, one in every thousand is found with the adult type of lesion. For every boy 2.8 girls are found. About five years later at least 24 per cent of these are dead, the girls showing double the mortality of the boys. Obviously, therefore, the most extensive and intensive application of known diagnostic, prophylactic, and therapeutic measures, is of the greatest importance in the problem of tuberculous infection and disease in children preceding this age.

As to prognosis and diagnosis in the individual case, numerous observers have pointed out the value of a study of the white blood cells. Hamil[27] made numerous blood examinations on sixty-eight patients who presented evidence of tuberculosis in varying degrees. He found that the monocytes are increased above the normal figures in active tuberculosis; that the total lymphocyte count is decreased below the normal figure in all active types of tuberculosis, and is increased in the healing stage; and that the ratio of monocytes to lymphocytes (M:L ratio) is increased in active tuberculosis, and decreases as healing occurs. These findings are corroborative of the reports of Sabin, Cunningham and Thompson, and Blackfan and Diamond. Friedman, Dameshek, and Hawes[21] conclude that a study of the peripheral blood and of the sedimentation rate of the red cells in suspected cases of hilum lymph node tuberculosis is of great interest and may prove to be of some practical importance.

The treatment of children with tuberculosis depends upon the seriousness of the tuberculous lesion. Opie[47] suggests the following classifications:

A. Demonstrable tuberculous infection with the following characteristics is prevalent among healthy children and requires no special care to prevent its further development:
   1. Infection revealed by the tuberculin test with no lesion demonstrable by roentgenologic examination.
   2. Circumscribed pulmonary nodules that have undergone calcification.
   3. Roentgenographically demonstrable tuberculosis of tracheobronchial lymph nodes, with the exception to be cited.

B. Children with the following conditions are in danger of tuberculosis that will undermine health and should receive special care in open-air schools, preventoriums, or by other means to prevent further progress of the disease.
   1. Latent tuberculous infiltration of the childhood type evident in roentgenograms, often associated with tuberculosis of tracheobronchial lymph nodes. In some instances, particularly in older children, these lesions are the scars of healed infection and do not require special care.
   2. Tuberculosis of tracheobronchial lymph nodes with some calcification, recognizable by roentgenologic examination (a) where the child is still in contact with open tuberculosis, (b) when the tuberculin reaction is intense, (c) when the lesions are unusually large or very numerous.

3. Latent tuberculosis of adult type.
4. Arrested pulmonary tuberculosis, notably in children who have been discharged from sanatoriums.
5. An active tuberculin reaction or an otherwise negligible tuberculous lesion recognized by roentgenologic examination in association with impaired health and conspicuous underweight, which are perhaps not referable to tuberculosis.

C. Children who should receive sanatorium treatment or its equivalent are:

1. Children with tuberculous lesions accompanied by symptoms and physical signs referable to tuberculosis.
2. Children with latent lesions of the lung either of childhood or adult type, that on repeated roentgenologic examination are found to be progressive.
3. Children with massive tuberculosis of tracheobronchial lymph nodes that has not undergone calcification.

But the physician's task is not completed when he has demonstrated a positive tuberculin reaction, determined the extent of the tuberculous process roentgenologically, and prescribed the necessary therapeutic course; it remains to determine the source of the child's infection and to institute such measures as are necessary to prevent future reinfection. Pope[50] concludes that exposure to open pulmonary tuberculosis in the household appears to be the most important single factor in the development of tuberculosis in children. Zacks[65] says when a case of tuberculosis comes under our observation, it is our responsibility to examine all the members of the family, physically and by roentgenogram. The significance of tuberculosis as a family disease has been stressed by many authors notably Myers, Opie, Chadwick, and McPhedran. Myers says that in a study of the spread of tuberculosis Lampson found that 67 per cent of the individuals in families where open cases of tuberculosis existed had been infected—this in contrast to 2.5 per cent incidence of infection in families where no tuberculosis existed. Opie and McPhedran have found that the infection spreads to practically 100 per cent of the members of families who are exposed over considerable periods of time.

Again Opie finds from studies carried on at the Henry Phipps Institute during the last six years, that at least 9 per cent of the children known to have been exposed to open tuberculosis in the family group develop clinically manifest disease; many develop latent infiltrating lesions of childhood or lesions of adult type, and tuberculosis of tracheobronchial lymph nodes is found in approximately one-fourth of them.

There can be little question, when one looks at the problem from the standpoint of tuberculosis only, that the ideal goal would be achieved if all children could be permanently removed from tuberculous households, but it is equally obvious that as yet there is neither financial, social, nor educational support which permits any such wholesale procedure. Dependence must be placed upon the closest sort of cooperation between public health officials and physicians, and upon the exercise of judgment and tact by these two groups in dealing with tuberculous families to the end that the environment may be made as safe as possible for the growing child.

Of the prophylactic measures employed in tuberculosis only one requires discussion. The BCG vaccination method of Calmette is still

the subject of considerable criticism. Calmette[9] states that since 1921, 392,000 infants and children in France and her colonies, and 120,000 in 30 other countries have been vaccinated with BCG. The general mortality rate for the vaccinated as given by Calmette is 8.6 per cent, and for the nonvaccinated 19.0 per cent. The mortality rate from tuberculosis for the vaccinated group is given as 2.6 per cent, and for the nonvaccinated group as 15.8 per cent. The accuracy of Calmette's statistical conclusions have been questioned.[25]

Another criticism of BCG which has been raised is that under certain conditions the supposedly avirulent culture may resume its virulency. Petroff and Brauch[49] report that they were able to grow two types of colonies from the original BCG culture. One, the "R" type, was nonpathogenic, and the other, the "S" type produced progressive disease in animals. Neufeldt in Berlin examined Petroff's cultures and contended that they were contaminated with a human strain of virulent organism.

Sasano and Medlar,[55] using a rabbit serum culture medium in place of the customary bile-glycerine-potato medium, found that the original nonvirulent BCG culture became definitely virulent for rabbits. They conclude that BCG is not a virus fixé, and that its stability depends entirely upon the environment. The catastrophy at Lubeck in which 67 infants out of 249 supposedly vaccinated with BCG lost their lives was a severe blow to the cause of BCG. There are many who believe that due to a change in the type of the culture media used by Professor Deycke, the BCG strain of avirulent organisms assumed virulency. Professor Lange after careful study and numerous experiments with organisms recovered from the lesions of the dead infants concludes that the tragedy was due to contamination of the BCG vaccine with human bacilli.

In this country Park[48] has carried on well-controlled experiments both in animals and human beings. He feels that an increased resistance comes from inoculations of BCG vaccine which roughly corresponds in time to the duration of the tuberculin reaction. On this basis he believes subcutaneous or intracutaneous inoculations are superior to oral administration, because longer lasting and more frequent positive tuberculin reactions are secured.

Heimbeck[28] reports some very interesting observations on the nurses of a tuberculosis ward of a hospital in Oslo, Norway. Von Pirquet tests were done on the entering classes from 1924 to 1927 with the result that 220 gave negative reactions and 200 gave positive reactions. In the course of a year or two practically all the negative reactors had become positive. Fifty-one of the nurses developed tuberculosis, only three of which were among the original positive reactors, the remaining 48 cases occurring in the negative reactors. He concluded, therefore, that a positive tuberculin test was an indication of immunity. In the entering class of 1927 were 23 nurses whose tuberculin reaction was negative; twelve of these were inoculated subcutaneously with BCG, the other eleven refusing. The result was that up to the time of the report (March 1928) "four of the eleven with negative reactions to the von Pirquet test who refused vaccination and who worked in the hospital among its tuberculous patients have a tuberculous dis-

case, but of the twelve who were vaccinated and whose reaction to the von Pirquet test in this way became positive, none has a tuberculous disease."

It is apparent that this viewpoint is in sharp contrast to the opinions of Rathbun, Stewart, and Myers previously referred to in this paper, to the effect that the adult type of tuberculosis occurs in conjunction with, or engrafted upon a preexisting childhood type of infection in a majority of the cases. If it be true that the childhood type of tuberculosis is benign and nonfatal, and the adult type is serious and killing, what is one to conclude as to the advisability of rendering the tissues of large numbers of children allergic to tuberculo-protein by BCG inoculations?

Further light needs to be shed upon the question of whether or not a positive tuberculin reaction is an indication of immunity, and whether or not BCG is safe and effective.

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American Academy of Pediatrics

Proceedings

FIRST AND SECOND ANNUAL MEETINGS OF THE AMERICAN ACADEMY OF PEDIATRICS

Report of Committee on Medical Education

AT THE time of the appointment of the Committee on Medical Education of the American Academy of Pediatrics, a committee of the White House Conference was at work on a detailed study of the same subject. It was obvious that it was waste effort for the Academy committee to consider the subject until this report appeared. Copies of the report of the White House Conference are now available and have been sent to each member of the Academy.*

Education in Pediatrics may be divided into three main topics:

I. Undergraduate instruction.
II. Postgraduate instruction.
III. The training of the Pediatrician.

The White House Conference report dealt only with the first two topics. Certain points were stressed in this report.

First: Pediatrics is a fundamental basic clinical subject and should be independent and of equal academic rank with other departments. In at least 11 schools today it is a subdepartment of medicine.

Second: Adequate facilities should be provided in the way of staff, hospital beds, and out-patient clinics, and the minimum requirements for satisfactory teaching were outlined. Many schools fall short in one or more of these facilities.

Third: 200 teaching hours should be the minimum assigned pediatrics in the medical school curriculum. Many schools fall short of this number. The better schools as a rule give this number or more.

Fourth: The report outlined the minimum course by topics, and recommended the study of pediatrics from a pedagogical standpoint.

Fifth: The 4 week intensive review courses, and state extension courses for general practitioners were found worthy of encouragement and continuation.

The White House Conference Committee did not consider in any way the question of the training of the pediatrician.

Your Committee has reached the following viewpoints for the present time:

1. That through its officers, committees and individual members the American Academy of Pediatrics should endeavor in every way to help in the carrying out of the recommendations of the Committee of the White House Conference. It urges that members should study the report and use their influence to improve

Presented to the American Academy of Pediatrics, Atlantic City, May, 1931.

The Committee consists of Drs. Borden Veeder, Chairman, T. B. Cooley, J. I. Durand, J. L. Gamble, Henry Helmholz, A. G. Mitchell, W. A. Mulherin, D. Lesesne Smith.

*Pediatrics: Education and Practice. Report of Committee on Medical Education. Section I, Medical Service. White House Conference. New York, Century Co., 1931.

the teaching of pediatrics in the medical schools from which they have graduated or with which they may be connected at the present time. Improvement can only be slow and gradual, but it will take place as the deans and heads of schools and departments become aware of the importance and scope of the field of pediatrics.

2. The Committee recommends that the Academy of Pediatrics continue the work of the White House Conference Committee by collecting and revising from year to year information relating to the teaching of pediatrics.

3. That the members of the Academy endeavor in their home states to develop and maintain extension courses, following in general the so-called North Carolina plan.

4. That it is the immediate function of the Academy of Pediatrics to study the subject of the training of the pediatrician.

In relation to this fourth point the Committee wishes at this time to make certain general statements.

In 1929 there were over 3500 physicians listed in the A. M. A. Directory as practicing pediatrics exclusively, or as their chief interest; 1489 were listed in the first group. The membership of the Academy is almost entirely made up from this group of physicians. This group who are styled pediatricians or pediatrists are a most heterogeneous mass, so far as their training and even their work is concerned. The vast majority are men in the practice of medicine. Some have simply developed by choice and opportunity into children's work with little or no special training, while others have spent years in training before entering upon practice. It includes men chiefly or solely interested in research work, but who are not in practice, and men whose *chief* or only work lies in school or child hygiene activities. It is a group which has grown by leaps and bounds in recent years and is increasing in almost a geometric rate of progression. Those who have been watching the movement expect to see this number more than doubled in the next few years.

The Committee feels that it is the function of the Academy to study this situation, to outline, if possible, minimum requirements in training, and to define standards. Perhaps, even, ultimately to find some way to enforce standards if this seems desirable and feasible.

One of the outstanding trends of American medicine in recent years is the development of specialism. While the reasons for this are interesting and important, and perhaps what has been gained in the field of medicine has in some ways been offset by equally important losses, we may accept this growth as accomplished and that is the important fact for the purpose of our discussion.

There are two methods by which a specialist may be defined and specialism regulated. One is by state or governmental control and regulation. This has been attempted in several European countries. The chief trouble with the United States today is that we have too much governmental regulation. If one stops for an instant to consider the difficulties to regulate the practice as medicine as a whole in the United States, the possibility of regulating the specialist becomes almost an absurdity. Whatever has been done of value in the raising of standards has been the result of tremendous effort on the part of the medical profession itself. When the government has entered in as a result of political or emotional reasons, measures have been introduced inimical to the best interests of the profession. No matter if there may be nothing but good in the thing itself, the medical profession should resist any and all governmental efforts to regulate the practice of medicine. It is a thing once started that has no end. In a developing, changing science, such as medicine, the interpretation of facts and the knowledge of one day is quite different from that of another, and one cannot make facts or knowledge by legislation.

Any worth while efforts to regulate specialism must come by the second method, that is, by efforts within the medical profession itself, and such regulation should be controlled by them. Such efforts are few and are only in their infancy.

Up to the present time the chief efforts have been made in surgery, obstetrics, ophthalmology, and in oto-laryngology. All in brief have attempted to regulate specialism by making a society membership the hall mark of a qualified specialist, and the passing of some form of an examination a necessary qualification for admission to the society. This follows in general the English method where a chartered society issues a degree, which in a small, old, compact country as Great Britain is quite generally recognized. In the United States where letters after a name and degrees are of relatively little importance, and often may be bought for a modicum of mental effort and training, plus a consideration, the use of new letters or degrees is rather an empty gesture and their chief use is the elevation of the individual's idea of his self importance. There is no legal or even moral right to limit specialism to members of these societies and it is quite probable that legislative attempts along this line would be unconstitutional. Their value and importance lie rather in the defining of standards which serve as a goal to which the young medical student and graduate desiring to specialize must be influenced and encouraged to gain.

There are many objectionable features to this "society method," but no other method of more promise has been offered. At the recent meeting of the American Medical Association a resolution was offered directing the Association to take up and study the question of the specialist. This is a matter that should be closely watched. It is probable that each group within itself is best qualified to define its own requirements of training. Most of us would resent and ridicule any attempt of surgeons or of ophthalmologists, for example, to define standards for pediatricians. Should the A. M. A. through its House of Delegates seriously undertake the defining of standards and control of the specialist, the pediatricians must be prepared to make a united and audible voice in regard to their own field of work. This we know cannot now be done through the Section of Pediatrics of the A. M. A., as it is limited to purely scientific subjects and has no permanent organization. No other pediatric society is in a position to express the pediatric viewpoint or speak for the pediatricians as a group. Even if there were no other reasons for its existence the Academy of Pediatrics has a function in speaking for the pediatricians as a group in such matters as education and public health.

In considering the training of the pediatrician in a specific way many difficult problems at once arise. First of all we must have a pretty clear definition of what we mean by pediatrics. To the average physician and to the laity it usually means something to do with sick children. If we examine the pediatric literature of recent years, the scope of our field, and the tendency of modern pediatric thought, a definition based upon such a conception is totally inadequate. But some definition of the field is necessary before we can discuss specific points and objective in education.

The Chairman of the Committee has attempted to make a definition, which is open to discussion. The definition has been submitted to a number of pediatricians who agree in general with the thought if not absolutely in the way it is expressed. The definition is as follows:

"Pediatrics is that branch of the medical sciences which has to do with the factors influencing the growth and development of individuals from birth to maturity."

Obviously much might be added in the way of clarification or elaboration. Thus growth and development includes both physical and mental growth. The factors

influencing growth could be expanded into their chief subdivisions, as heredity, disease, and environment. The latter used in a broad sense can be subdivided ad infinitum, as it includes a multitude of factors which influence the development of the individual.

The first criticism of the definition is that it is too broad. Unfortunately from the standpoint of defining standards and qualifications for use of the term pediatrician or pediatrist, the subject of pediatrics is too broad. The field of pediatrics is so broad and fundamental that it differs from the specialties. It has, as has so often been pointed out, its center in the child, and is not based on some anatomical system, disease process, or special technic. Some one of these three things forms the basis for the specialties. It is comparatively easy to set up standards and qualifications for the specialties, but exceedingly difficult for pediatrics.

As the Committee sees it, this is a task which will involve much thought and work, and the integration of many viewpoints. Your Committee feels that all it can do at this time is to indicate the field of work for the Committee on Education and point out some of the inherent difficulties.

The Committee recommends that the following reply be made to the Follow-Up Committee of Section 1 of the White House Conference, who has submitted their report on Pediatric Education to the Academy of Pediatrics.

1. That the Academy endorses the report and recommendations.

2. That the Academy will endeavor through its officers, committees, and individual members to help in carrying out the recommendations.

3. That through its regional and state groups it will encourage and aid in the development and maintenance of extension courses for postgraduates.

4. That it requests the White House Conference to turn over to it the data and material collected in its study of pediatric education. The Academy will for a period of years keep this data up to date by a yearly survey of changes in the facilities for teaching, details of instruction, etc.

Motion made, seconded, and carried that these recommendations be adopted.

The Necessary Training for a Pediatrician
Report of Committee on Pediatric Education

Borden S. Veeder, Chairman

THE present work of the Committee on Education of the American Academy of Pediatrics is in a sense a continuation or extension of the study of pediatric education made by the White House Conference. The White House Conference report did not deal with pediatrics as a restricted field of the practice of clinical medicine, as this aspect of pediatric education and practice did not come within the scope of its interests. The Academy of Pediatrics on the other hand has a direct interest in this subject as its membership is made up of physicians who limit their field.

In the report made at the Academy meeting at Atlantic City in 1931, it was stated that the Committee would (1) continue and keep up to date the material gathered by the White House Conference, and (2) study the question of restricted practice. As a result of the adoption of this report a request was made of the Follow-Up Committee of the White House Conference for the material they had gathered, and this has been turned over to the Academy Committee with the understanding that the educational studies will be continued by the Academy. A study

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of the utilization of undergraduate teaching hours is being made and will be published later in THE JOURNAL OF PEDIATRICS.

In this report the Committtee wishes to present the material so far obtained in regard to the question of restriction of practice to the field of pediatrics. In other words, in regard to specialization: although for reasons given in the report last year, we object to the use of the term "specialist" in referring to pediatrics.

A consideration of the subject leads to certain conclusions.

1. There has been a rapid increase in recent years in the number of men who are limiting their field to pediatrics and who call themselves pediatricians.

2. Special training is necessary for the men who limit themselves to this field from both professional and ethical viewpoints.

3. There are no legal or professional limitations or restrictions upon those entering this field.

4. There are no professional standards which can be used to define minimum or even ideal educational requirements.

5. Any attempt to impose ethical, professional, or legal restrictions upon those limiting their field to pediatrics must necessarily be based upon standards which at the present time are nonexistent.

Whether the ultimate solution of the question of specialization depends on legal restrictions, or on professional restriction governed by some national board, or indirectly through the prestige of some organization such as the Academy of Pediatrics, some definite educational standards must form the basis upon which restriction or limitation is made.

The subject is intricate and involved. It is the feeling of the Committee that no matter how the question is ultimately solved, the basic and fundamental need is for a more or less concrete or specific idea of the special training which is necessary for a physician to limit his practice to the field of pediatrics. It is the purpose of the Committee to attempt to gather the material together upon which standards may be based. It may be stated definitely at this point that the Committee at the present time presents no conclusions or recommendations. It will simply try to present what has been done so far in studying the subject. The way in which to approach the subject was the first problem considered. There is no factual data upon which conclusions can be based. It eventually simmers down to a matter of opinion. Paralleling the rapid growth and changes in pediatrics which have taken place in recent years we find that pediatricians have received their training in a multitude of ways. The training and education of the men entering the field today is quite different from the training and education of the men graduating 15 or 20 years ago, as facilities and opportunities for training in pediatrics have increased by leaps and bounds in the United States. Discussion with a number of pediatricians showed that considerable difference of opinion exists in regard to what constitutes the best or ideal method of training.

It was finally decided to obtain the personal opinions of a large group of pediatricians and find if some general factors entered in upon which all or a majority agreed or could agree. To obtain this information a brief questionnaire was sent to every member of the Academy in January, 1931. Two hundred ninety-three replies were received by the end of four weeks when the polls were closed and a number of subsequent replies have not been included. In order to obtain definite specific replies which could be summarized, and to be certain that the same subject was being considered by all, the question was asked as to what the individual considered the *minimum training* a graduate of a class A medical school should have before limiting his field to pediatrics. There was nothing in the questionnaire which stated or even implied that this opinion had anything to do with the requirements for admission to the Academy of Pediatrics. Nevertheless the

Committee received numerous dissertations on the subject. The Committee on Education has nothing to do with this subject and it is not included in the scope of their field or function. That such data as they present may ultimately be found of use for such a purpose is of course within the realm of possibility. We wish to emphasize this statement so that discussion of the report will not go over into fields which are not within its province. As stated above the Committee is making no recommendations at the present time—it is simply studying the subject. It would seem logical to believe that there must be some essential minimum amount of training necessary before a physician should limit his field to pediatrics. That such a minimum should be an advisable standard is not suggested.

We wish to present the data obtained in regard to the opinion as to minimum requirements. Twenty-three of the 293 replies to the question of minimum requirements could not be tabulated for one reason or another. This left 270 replies upon which the tables which follow are based. Figures are given only in percentages for the sake of simplicity. As the replies were received they were divided into three groups A, B, and C based on the year of graduation. The replies were tabulated in this way as follows:

GROUP	BASIS	RECEIVED	NOT USED	TOTAL USED	
A	Graduated before 1910	105	8	97	A
B	Graduated 1910-1919	137	12	125	B
C	Graduated 1920-1925	51	3	48	C
		293	23	270	

This grouping into A, B, and C will be used in the succeeding tables without further discussion. Percentages are based on the last or "total" column of 270, or groups A, B, and C, unless otherwise stated, as we are concerned with positive rather than negative findings. Fractions have been rounded.

Service in a General Hospital

Table 1

General Hospital Training

1 Year	80%
1½ Years	8%
2 Years	3%
Less than 1 Year	3%
Definitely stating "unnecessary"	3%

Showing the frequency service in a General Hospital was considered as a minimum requirement by percentages according to length of service advised.

It is apparent that the vast majority of pediatricians consider a year of general hospital training necessary before entering upon the specialized study of pediatrics. There was no important difference among the three groups in this regard. Almost without exception the men stating definitely that general hospital training was not necessary, indicated training in a children's hospital for two or more years. The comment was made by them in one way or another that the modern large children's hospital gives an all around service (surgery, nose and throat, etc.) and hence a general hospital training is no longer necessary.

When we find that 91 per cent consider a year, or even longer, of general hospital training indicated, and those regarding such a service as unnecessary have a qualified children's hospital in mind, we must regard the opinion in this matter as almost unanimous.

Service in a Children's Hospital

Service in a children's hospital was regarded almost without exception as a necessary minimum requirement. Considerable difference of opinion exists as to the length of service. This is shown in Table 2.

Table 2

Service in a Children's Hospital

1 Year	59%	A 63% B 60% C 46%
1½ Years	3%	
2 Years	32%	A 29% B 30% C 44%
3 Years	3%	
Less than 1 Year	2%	

Showing the frequency service in a children's hospital was considered as a minimum requirement, by percentages according to length of service advised, and by groups.

While we find a unanimity of opinion in regard to training in a children's hospital being a minimum requirement, there is approximately a 60:40 ratio between the group regarding one year as the minimum and the group regarding a longer time as necessary.

An interesting viewpoint is shown if we consider the 1 and 2 year recommendation from the time of graduation of the person making the reply. It will be noted the ratio between 1 and 2 years for Groups A and B is a 2:1 ratio in considering 1 year as sufficient for a minimum. The ratio of Group C is a 1:1 ratio. In other words half of the more recent graduates feel 2 or more years of training in a children's hospital should be a minimum requirement, as contrasted with only a third of the older graduates.

Infectious Diseases

Approximately three-fourths of the replies stated that training in a hospital for contagious diseases should be among the minimum requirements, as is shown in Table 3. Less than 2 per cent made a definite statement that they considered this unnecessary.

Table 3

Service in a Hospital for Infectious Diseases

1-3 months	23%
3-6 months	27%
Over 6 months	5%
As part of service in children's hospital	18%

Showing frequency and length service in an infectious disease hospital was considered as a minimum requirement, by percentages.

Thirty-five per cent of Group C who, as has been pointed out, tend to favor a two year service in a children's hospital, think that the service in an infectious disease hospital should be included in the children's hospital service.

Out-Patient and Infant Welfare Work

The recommendations for these two subjects as a minimum requirement tally so closely they may be considered together. Seventy-one per cent of the replies in-

cluded such training. There is a distinct difference of opinion, however, as to whether such work should be an integral part of the hospital service or should be taken up after the individual has entered practice. This is shown in Table 4.

TABLE 4

SERVICE IN CLINICS

Out-patient clinic:		
As part of hospital service	26%	A 23% C 40%
While in practice	46%	A 53% B 31%
Well Baby Conference:		
As part of hospital service	26%	A same as C above.
While in practice	45%	

Showing service in out-patient clinic was considered as minimum requirement, by percentages, and according to time when service should be taken.

This table again shows the difference in group thought between the older and more recent graduates. The younger men tend to favor a longer children's hospital service, including the out-patient work in this service. This perhaps reflects the improvement in hospital service in recent years.

GENERAL PRACTICE

In this connection we had to distinguish between "general practice" and "private practice in pediatrics." The latter was included by a number of men who were considering requirements for admission to the Academy. A great many men, particularly among the group graduating over 10 to 15 years ago, have entered the field of pediatrics via the route of general practice. A large group of the more recent graduates have entered the field directly from their service in a children's hospital. The thought that general practice should be a minimum requirement clearly reflects this difference. Taken as a whole 25 per cent consider that experience in general practice should be one of the minimum requirements. By groups the percentages are as follows:

A 30%
B 20%
C 10%

Nine per cent state specifically that it is not necessary. Another 10 per cent of Group A speak of it as being desirable but not necessary. None of Group C had the same feeling.

About 60 per cent of the total did not mention general practice and it may be inferred that they did not consider it necessary. As a whole then we may conclude that a large majority of pediatricians do not consider general practice as an essential minimum requirement or method of training.

The length of time suggested for general practice was as follows:

1 year 3.5 per cent
2 years 9 " "
3 years 12 " "

By groups, the 3 year recommendation was as follows:

A—26%
B— 6.4%
C— 0%

Assistant to an Established Pediatrician

Nine per cent of the total advocate from 1 to 3 years of assistantship as a most valuable method of training, although not including it as a requirement.

Length of Time of Training

From the various methods of training advocated we turn to the question of the period of time necessary to complete a minimum training to limit the field to pediatric practice. It was possible to calculate the time which the minimum requirements suggested in 228 replies would take to complete. The percentages in the following table are based on these 228 replies, rather than on the total 270 used in the previous studies. This data is shown in Table 5.

Table 5

Time Required for Minimum Training

	A		B		C		TOTAL	
1 year	0		1		1		2	13%
2 years	4		16		8		28	
2½ years	14	52%	22	64%	8	60%	44	
3 years	17		34		10		61	60%
3½ years	10		12		7		29	
4 years	10	43%	11	20%	6	20%	27	27%
5 years or more	24		10		3		37	
	79		106		43		228	

Showing the number of years of necessary training required to complete the minimum requirements by groups for periods in years, and percentages for certain groupings.

A glance at this table shows that as a whole Group A favors a longer period of time than the B and C Groups. Thus 43 per cent of A considers 4 or more years necessary as compared with 20 per cent of B and C. This difference is due to the large number of the A Group who advocate 2 to 3 years of practice as one of the minimum requirements during which time out-patient work is being done.

We note that 13 per cent outlined a minimum training of 2 years or less: 60 per cent a training of from 2½ to 3½ years: 27 per cent a training of 4 or more years. The mean lies between 3 and 3½ years. Twenty-seven per cent outline a training receiving 3 years, 33 per cent a training of less than 3 years, and 40 per cent a training of more than 3 years. The conclusion that 3 years of training after graduation is the necessary length of time which must be spent to acquire the minimum training which would enable a man to limit his field of work to pediatrics, represents perhaps an average opinion.

Without any desire to draw conclusions or make recommendations, the committee has attempted to make from these figures and the comments in the questionnaire a picture of what might be called an average of opinion. While some undoubtedly will feel the training outlined as too short, others will consider it unnecessarily prolonged, as it represents an average of personal opinions.

The average opinion of the minimum amount of training a graduate of a class A medical school should have to limit his field to the practice of pediatrics and to be considered as a pediatrician is about as follows:

I. A period of training of approximately 3 years.
II. The first year should be spent as an interne in a general hospital.
III. The remaining two years should be devoted to definite pediatric study. It should be utilized in one of two ways.

1. Two years' work in a children's hospital, which includes service in a ward or unit for contagious diseases, and in the out-patient clinic which has a well baby service as well as ambulatory sick.

2. One year's service in a children's hospital followed by a year spent in out-patient and well baby clinics and a 3 months service in a hospital for infectious diseases.

IV. Following this direct training a physician may be justified to start practice as a pediatrician. He should, however, for the next few years serve regularly in some capacity in connection with work for children, as hospital, out-patient service, public health clinic, in school hygiene, or in some other child caring institution or activity.

The discussion under "Remarks" in the questionnaire is of considerable interest. It is unfortunate that this cannot be tabulated or reproduced in toto. Certain opinions were expressed in one way or another in a number of replies which can be grouped.

A number of men referred to the character, quality and attitude of the hospital staff as of more importance than any other factor. As one man strikingly put it, "Six months service in one children's hospital would be worth more than 10 years in some other." Another excerpt with the same thought, "The value of the training is by whom given."

A number of the replies referred to the importance of the individual. "The character, mental capacity, energy, etc., of the individual is important." In other words, one man might receive an adequate training in much less time than another. The committee is fully aware that graduation from a Class A medical school and the receipt of an M.D. degree does not necessarily make a man a good or even competent doctor. The vast majority with this training, however, will have average competency. We further know of no way in which individual capacities can be foreseen, tabulated, or used as a standard. Many seemingly brilliant promises have faltered, and many an unpromising prospect has earned distinction.

Three per cent of the replies, chiefly from the younger group, suggested training in mental hygiene and child psychology as an essential subject of training. A slightly smaller number referred to the importance of public health work in one form or another.

That training in the handling of the normal child is essential was mentioned in one way or another a number of times. A few men mentioned laboratory or research work as an essential part of the training.

Many men—perhaps half—indicated in one way or another that they did not consider "minimum requirements" would make a pediatrician; that a man must go on working with children for years to make himself a competent, thorough pediatrician. With this viewpoint the committee thoroughly agrees.

As previously stated, many men worked themselves into a viewpoint that the committee was endeavoring to establish minimum standards on requirements for admission to membership in the Academy of Pediatrics. Many gave what they considered as "equivalents," as years of practice, etc. While these were of interest they simply confused the subject under study and so have been disregarded. They refer to the past rather than the present or future.

Short postgraduate courses were mentioned a number of times as an example of insufficient training. The course of study outlined for minimum requirements is a course of "graduate" study. We know of no short courses given which are intended to train men to limit their field to pediatrics, although it is certainly true that some physicians have taken postgraduate courses and announced themselves as pediatricians. We cannot hold with the view expressed "That 'postgraduate'

courses ought to be stopped'' for this reason, nor can we see the possibility of complying with the request that ''the Academy should stop this.''

The committee does not wish to give the impression that the minimum requirements as outlined are presented as the way in which a pediatrician should receive his training. Much more complete and satisfactory training is desirable. The ''irreducible minimum'' is not necessarily the ideal or best method of education.

''IDEAL'' TRAINING OR REQUIREMENTS

Minimum requirements may be and usually are quite different from ''ideal'' requirements—a fact that was frequently expressed in the replies to the questionnaires. This question was not included intentionally in order to avoid a confusion in the answers. Before the replies to the first questionnaire were tabulated a second letter was sent to fifty pediatricians selected at random from those replying to the first, and without reference to the nature of their reply. This was not in the form of a questionnaire, but a letter reply was requested to the question as to what advice they would give to a graduate of a Class A medical school who planned to study and limit his practice to pediatrics. Age and financial status was not to be taken into consideration.

Forty replies were received. Approximately half advised a three year training (the time found later to express an average opinion of minimum requirements). Only one considered two years as sufficient. The other half of the replies outlined a course of study or preparation of four or more years.

A year of general hospital service was advised by all but one, followed by two years of work in a hospital for children. Those adding a fourth year urged another or third year in a children's hospital, travel, assistantship to an established pediatrician with time spent in welfare and out-patient clinics, investigative work and public health work (preventive?) with children.

The opinion was general that the service in a children's hospital should include during the two or three years, as the case might be, out-patient work, a well baby service, laboratory work and a contagious disease service.

Perhaps more important than the question of time were some of the ideas expressed. Some stated that more than 3 years of training in hospital tended to institutionalism and a wrong viewpoint toward the ''practice'' of pediatrics—the point we are discussing in this report.

The view that the fourth year should be spent in rounding out deficiences of the first three years and in travel, special study, child guidance work, etc., rather than in a hospital was along this same line. A point frequently emphasized was that the time spent in hospital training shall be in two or more clinics or hospitals and preferably in different parts of the country. Among those advising travel, several mentioned Europe, but not a single suggestion as to definite study in a European clinic was made. Nothing could be more expressive of the development of American pediatric clinics and hospitals which has taken place in the last few years.

A specific ''graduate course'' of training was advised but once. This brings up a point that has considerable theoretical interest. There seems to be a distinct feeling against a formal course of graduate instruction as a method of training for pediatrics. The view generally expressed is that living in a hospital, working in the wards and out-patient departments, granted that this is done under competent chiefs, is more valuable than formal instruction in ward classes and lectures.

There are but two ''graduate'' courses in pediatrics. One is associated with The Mayo Clinic and Foundation. This graduate course of three years compares closely with the training given at most of the larger pediatric clinics—much more so than with a formal course. The Graduate School of the University of Pennsylvania

offers a three year graduate course. After a year of formal instruction, the remainder of the time is spent in hospital training and investigative work. A study of their catalogues shows that practically all of the graduate students in pediatrics have taken only one—the first year. It would seem that this might be the best method of training for a physician who has been in practice for a number of years and who desires to limit his field to pediatrics. Practically all teachers and pediatricians with whom this subject has been discussed are of the opinion that hospital training (including laboratory and out-patient work) is preferable for the physician who enters on his specific training immediately following his medical school graduation.

An average view of an "Ideal Training" is roughly as follows:

I. Graduation from a "Grade A" medical school.

II. 1 year general hospital training.

III. 2 years Children's hospital service.

This should include: Clinical work in wards.
Out-patient work.
Well baby clinic.
Contagious disease service.
Preventive pediatrics.
Child Guidance work.

IV. 1 year Special Work in a Children's Hospital or Clinic.

(1) Completing gaps in previous 2 years' work.
(2) Investigation.
(3) Further clinical study.
(4) Travel.

Not more than two years of the last three should be spent in any one institution or under same chiefs.

As stated in the first part of the report the committee is not attempting to fix standards. It has simply tried to obtain and express in a more or less definite way the average of opinion and viewpoint regarding a matter which in its very nature is not factual. A subject which contains so many intangible variables, as the quality, character, personality, and intelligence of the individual, and the character, spirit, quality, and resources of the hospitals and their heads, is impossible of being reduced to fixed standards.

News and Notes

Syracuse University in connection with the **Central New York State Pediatric Club** will entertain and conduct a Clinic Day early in November. The meeting will consist of a series of papers and interesting case presentations. This club was organized several years ago following a Clinic Day given at the University of Rochester. Members are connected with the various medical colleges in Toronto, Montreal, Kingston, Albany, Rochester, Buffalo and Syracuse.

The Utah White House Conference for Child Health and Protection which was held in April, 1931, was one of the most successful ones held in the Union with 4000 interested people attending. Governor George H. Dern then appointed a continuation committee, the Utah State Council on Child Health and Protection, which was to continue the work planned at the White House Conference. This State Council has spent the past year in organizing County Councils which are to be units of the State Council. So far more than one-half of the Counties have been organized and in the near future they are certain that all units will have been organized for the plan has met with general approval by the people of the State.

At the time of the **Southern Medical Association** meeting in Birmingham, Alabama on November 16 and 17, there will be a meeting of members of the **American Academy of Pediatrics** in Region II. This will be held under the direction of Regional Chairman, Edward C. Mitchell of Memphis. This meeting will be held the night of November 15. It is planned to have all the commissioners of health in the states comprising Region II at a special meeting at this same time. The purpose of this is to bring together more closely the state chairmen, the regional board and the commissioners of health for a closer cooperation in the various phases of child welfare.

Erratum

In the Abt and Traisman article in the August issue, p. 181, line 14 should read: "The third week, 0.015 gm." instead of 0.115 gm.

Book Reviews

Behavior Aspects of Child Conduct. Esther L. Richards, M.D., D.Sc., Macmillan Co., New York, 1932, pp. 299.

This book should be read by every physician who is interested in children. In these days of specialized child psychology fads and fancies and educational experiments which propose to change the nature of child behavior and development in some wonderful but mysterious way, it is a welcome breath of sane, broad, and practical discussion.

The book has its basis on a series of lectures to teachers, parents, and physicians, and hence lacks the formality and dryness of a textbook. It is most readable. The first nine chapters discuss the influence of environment, physical handicaps, school and intellectual difficulties, social conditions, early habit training, constitutional endowment, and play on behavior. In these chapters Dr. Richards draws a background of the necessity of a thorough systematic approach to the study of the individual child with a problem. The theme that lies back of the discussion and which the various chapters bring out is:

1. Mind is the individual in action; its expression involves the coordinated activity of every cell in the organism.

2. Our aim should be to interpret rather than to judge conduct.

3. The interpretation of behavior involves a study of the individual intellectual ability, physical status, habit training, social and economic conditions, social contacts, play outlets and constitutional endowment of emotional control.

The latter part of the book contains chapters on the fears of childhood, the delinquent, the management of adolescence and a final word on community consciousness. Throughout the book the points the author stresses are illustrated by pertinent case histories.

One of the most important and outstanding factors which tends to put this book in a niche by itself among the multitude of books on child behavior, is the feeling that in the discussion of the child the author is either consciously or subconsciously relating the childhood problem to the total life of the individual. This arises from the fact that the author by her work and training has not confined her work to children. It is a much needed perspective in the field of child behavior for distinguishing between the essential and nonessential.

Dr. Richards' book will go a long way in the integration of psychiatry and child psychology with pediatrics. The two fields are slowly but surely finding their close relationship. The matters which this book discusses are only superficially, if at all, taught in our medical schools and we can say without reservation that a reading will broaden the viewpoint of every physician in the field of pediatrics.

BORDEN S. VEEDER.

The School Health Program. White House Conference on Child Health and Protection, The Century Co., New York, 1932.

This publication may be read from two essentially different viewpoints, i.e.:

One essentially educational and the other medical. The educational deals with knowledge, skills and attitudes favorable to health. The medical deals with sound-

ness of the knowledge to be imparted and the actual professional service rendered.

The educational viewpoint was dominant in the Committee, hence the report is essentially the expression of that viewpoint.

The book is prefaced with a scholarly presentation of A Philosophy of Education, by William H. Kilpatrick, Ph.D., Professor of Education, Teachers College, Columbia University, New York City, which will aid the physician who reads it to better understand the aims of modern education and the philosophy underlying these aims.

The survey summary shows a bird's-eye view of "What is now being done" from a quantitative consideration. The physician would be much more interested in the quality of the medical service given in schools, especially in the physical examination given the child upon his entrance to school. This is not discussed in this publication. The construction and operation of the school plant are discussed from the standpoint of health and safety and the advantage of the combined or consolidated district school in rural areas is easily appreciated from the health standpoint. The physician unfamiliar with modern planning will get a good idea from this chapter.

Mental hygiene, whether one views it as an old or new development, is finding more favorable consideration in educational plans but as yet the subject is probably more extensively discussed than practiced as a regular part of the school health program in the detail recommended in this report.

Physicians will no doubt be surprised to find medical service dismissed in half the number of pages devoted to dental service, or to nursing service, but the report of the physicians in this Committee is concisely presented and in familiar language.

The medical Sub-committee in their report to the Chairman of the Committee on The School Child, endeavored to establish the following points:

1. The medical inspection now carried on is but a rough screening process for signs and symptoms of communicable disease or obvious handicapping physical defects.

   Such an inspection is *not* a "periodic health examination" as the term is used in a medical sense.

2. The aims of school medical service are preventive and conservative; and curative services are the function of agencies outside the school system since the school was founded for education and all its activities must be justified on this basis.

3. Superior facilities for medical treatment are provided elsewhere in the community by agencies founded primarily for health protection and restoration.

4. A valid record of the physical assets and liabilities of each pupil and each of the school personnel should be kept in each school to allow for adaptation of the educational program to individual needs in the handicapped.

5. The educational and administrative programs should be elastic enough to make adequate provision for the physically and mentally classified pupils.

6. Medical authorities are the best judges of the soundness of the health information to be taught.

7. School physicians should qualify first as good physicians by showing good clinical judgment. He must and will learn to fit into the school program effectively and with as little undue disturbance of the program as possible. Staff medical inspectors tend to lose interest in the routine dis-

charge of their duties and should then be replaced. This improves the work in the schools and provides understanding and better cooperation with the medical practitioners many of whom have had experience in the school health work.

8. Greater interest and cooperation should be fostered in the medical societies so that the medical profession will aid the educational administrator in the selection of his inspectors and his choice of health material for teaching.

Dental, nursing and nutritional services must be closely integrated with the medical service and the physician will better understand the work done in school under these heads if he will read over the pages in this book which are devoted to these subjects.

The educational aims and methods used in the various intelligence levels may present some difficulties to the physician in view of terminology characteristic of educational groups, but whether he be in agreement or no, every physician should at least be familiar with modern health education efforts in the schools—public and private which includes the parochial and special schools for the Indian and Negro.

The physician is an influential factor in home and school cooperation perhaps not sufficiently appreciated in his private office capacity as in his hospital and clinical services to school children. He is vitally interested in school administration whereby the health interests of the child are kept paramount.

The physician will applaud the efforts recommended to require and furnish a better basis for health appreciation on the part of the teacher so that she may consider more fully the physical needs as well as the mental needs for the child.

Sound legislation should be preceded by careful experimental tryouts to see that it works. Opinions expressed by "44 out of 48 physicians" is significant only if the physicians are truly representative of medical thought. One might suspect from some of the recommended provisions of the school law suggested in the chapter on Legislation that the medical opinion tended to be selective.

LE ROY A. WILKES.

Comments

THE interest in pediatric education of the last few years may be looked upon as a sort of inventory or "stock-taking." That it is a subject of more than American interest is shown by its being made a topic for discussion at the last meeting of the British Pediatric Association and by a number of editorials in British medical journals. One of these discussed in considerable detail the report of the White House Conference.*

Pediatrics in recent years has not only developed by leaps and bounds as a subject of the medical curriculum, as evidenced by the development of many first-class university clinics and affiliated hospitals, but as a field of medical practice the growth has been even more remarkable. The academic growth may even be regarded as a response to the "field" demand. This is certainly true so far as funds and equipment, which may be regarded as necessary tools, have been made available.

Unlike, for example, the growth of bacteriology among the medical sciences, the development of pediatrics has not been due to new or wonderful discoveries in the field of science which have changed the conceptions of disease. Its growth rather has been a part of and paralleled the recent era of child consciousness which has been an outstanding trend in the social development of the past twenty-five years. That an increased interest in the health and physical growth of the child should take place during this time when the child has been placed in the limelight as a social unit is only natural. Perhaps the most fundamental truth or knowledge that has come out of it all is the interdependence of all phases and aspects of child life; the social, economic, intellectual, educational, psychological and physical influences and problems of the child are so intimately interrelated that they cannot be dissected out and treated as single entities. It is for this reason that what we are doing and teaching in regard to pediatrics in our medical schools is a matter of interest and importance. A conception of pediatrics limiting it to the diseases of childhood would make a university clinic thirty years obsolete, regardless of whether or not the hospitals and laboratories had been built in the last five years.

That the "claims and demands" of the White House Conference Report were not regarded by the medical schools and the medical profession as the yap of a group of "specialists" shows how definitely the position and scope of pediatrics has progressed in the last decade. This formulated statement of an existing condition which has gradually and insidiously developed in recent years was quite generally accepted. The following resolutions adopted by the American College of Physicians—the internist group—represents the attitude of the other clinical branches toward pediatrics.

> "The Committee on Public Relations has considered the Report of the Sub-Committee on Medical Education of the Committee on Medical Care for Children of the White House Conference on Child Health and Protection presented at the meeting of the section on medical service at Washington, D. C., February 20, 1931.
>
> The report deals extensively with the subject of Pediatric Education. Your committee recommends that the American College of Physicians heartily endorse the recommendations embodied in the report of the Sub-Committee on Medical Education, and express its appreciation for this able work in the interest of Pediatric Education."

*British Medical Journal, May, 1932.

THE present problem which is facing the Pediatric fraternity is one of a different nature. It has nothing to do with the undergraduate teaching of the subject and its place, field and scope in the general scheme of medical education. It is, one might say, a personal problem, as it has to do with the qualifications and training of the physician who limits his field to children. This same rapid growth in recent years which has been so important in developing the field and the subject, is the factor which makes the outlining of qualifications so difficult. The older men in the field today are to a very large extent self-taught. They entered by the route of general practice and specialized because of some inherent urge or opportunity of practice. The recent graduates who have entered the field have had far greater and better opportunities for intensive training. Whether in the long run the average will be better or worse than the average of the older group time alone will tell. With the present opportunities the average should be higher. The unknown factor is of course the average of the individual and his ability and intelligence to work.

The differences of viewpoint of what constitutes a satisfactory or necessary training are well shown in the report of the Academy Committee on Medical Education which is published in this issue of the Journal. It is a subject about which ideas rather than facts must be used. Time alone will show whether the conclusions drawn are correct.

The question has been asked as to why bother about qualifications for specialization and such matters. Why not let them take care of themselves as they have in the past? The rapid change in the character and type of American medical practice in recent years is in large measure responsible for this problem. Whether or not this will change again following the economic and social changes which have been taking place since the fall of 1929, as some believe, is a matter of speculation.

With the rapid growth of specialization a distinct problem has arisen in recent years which has been widely discussed and pondered over by educators and more directly by those responsible for medical licensure. Several of the State Boards of Examiners have definitely made plans for the licensing of "specialists" in their respective states. Such a specialist license must necessarily depend on "qualifications." Forty odd states making their own qualifications and requirements present a picture of chaos and complication beyond end. For the time being these are in abeyance while the matter is being discussed and pondered over by the Council on Education of the American Medical Association and the National Board of Examiners. It is beyond question that any recommendations of the American Academy of Pediatrics, based necessarily on a thorough study of the subject, will be the basis upon which future rules and regulations relating to the practice of pediatrics as a limited field will be made. We can either sit passively by at the present time, and vent our fury and spleen at some future time trying to change conditions, or as a group we can calmly and carefully study the matter and be quite confident that our decisions will be the basis for licensing Boards' rules and regulations if they are imposed at some future time. It is a subject that involves us whether we like it or not. It is one of the most important subjects involving the future of pediatrics.

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